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Soft Drink Consumption Among Yup'ik Eskimo Teenagers

by Maureen C. Thiele, M.P.H., RD[1]
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ABSTRACT

Soft drink intake among Yup'ik Eskimo teenagers was assessed as part of a monitoring system for a nutrition education intervention project. Soft drink intakes were found to be three to four times greater than in the general US teenage population. Over half of the soft drinks consumed were in the form of sweetened non-carbonated beverages, e.g., Tang, Koolaid. At the end of two years of intervention, soft drink consumption decreased by 10% in the 10 intervention villages as a whole, while intakes decreased by 4% in the 4 control villages. Soda pop consumption alone decreased by 18% in the intervention villages and increased by 20% in control villages.

INTRODUCTION

Excessive soft drink and candy consumption among teenagers is of concern to parents and health professionals because of their potential contribution to dental caries and poor dietary habits (1-3). Data from the Nationwide Food Consumption Survey, 1977-78 revealed that boys and girls, ages 13-18 years consumed an average of 221 gms of soft drinks/user/day during the winter season (4,5). Soft drinks consisted of carbonated beverages and non-carbonated beverages made from mixes or liquid concentrates. From nutrition surveys conducted in 1968 in Wainwright, Alaska, Sauberlich et. al. reported the importation of 14,400 cans of soda pop to this village of approximately 400 people (6). In 1977, Petersen and Bellinger reported that 211,000 cans of soda pop were imported that year (7).

The purpose of this article is to summarize the soft drink consumption patterns of Yup'ik Eskimo teenagers and to report on the changes that have occurred in villages where nutrition intervention activities were implemented.

METHODS

During November to May, 1985 and 1987, dietary intakes were assessed on teenagers living in 14 villages located in the Yukon-Kuskokwim Delta of southwestern Alaska. The purpose of these surveys was to monitor teenagers' dietary intakes before and after implementation of a three year nutrition intervention project. The 14 villages were divided into three groups. Groups one and two were designated as intervention villages and group three was our control group. Intervention activities began in Group one (4 villages) in 1985, and Group two (6 villages) began receiving services in 1986. Our control Group three (4 villages) did not receive services until after the end of our final data collection in 1987.

Nutrition intervention consisted of classroom presentations, consultation to school lunch programs, development and demonstration of culturally and regionally relevant nutrition education resources, and provision of nutrition consultation to other agencies and programs serving the target communities. The emphasis of education was on the importance of good nutrition to health, the potential harm of high amounts of sugar in our diets and the importance of traditional foods to health and survival.

Dietary data from 374 teenagers in 1985 and 563 teenagers in 1987 were collected using a twenty-four hour recall interview.

Teenagers in grades seven to twelve were interviewed by trained bilingual staff of the Yukon-Kuskokwim Health Corporation Nutrition Improvement Project. During the 1987 data collection period, an attempt was made to interview the same students as in 1985, but graduation or busy school and extracurricular schedules did not allow us to interview the same students. All students in grades 7-12 who were willing or available were interviewed at each data collection period. Interviewers used an adaptation of a method developed by Jean Hankin (8). Photographs of 23 pre-weighed foods, pre-portioned into typical small, medium and large servings were used to assist the interviewee in remembering portion sizes. Other aids included rulers, cups and plates. Respondents were also asked to draw odd-shaped food items and to provide dimensions in inches.

RESULTS AND DISCUSSION

The amount of soft drinks and soda pop consumed during a 24-hour period by the teenagers are listed in Table 1. Soft drink intakes in all villages averaged 25 ounces per day in 1985 and decreased to 23 ounces per day in 1987. If we assume that soft drink intake remains constant throughout the year, the annual per capita intake for 1987 would have been 1,023 cups. Overall soda pop consumption averaged 9 ounces per day in 1985 and decreased to 7 ounces per day in 1987. If consumption patterns were maintained throughout the year, annual

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Table 1. Mean Daily Consumption of Soft Drinks Among Eskimo Teenagers by Village Groups Grams (ounces)

Village Group	Soft drinks+ 1985	Soft drinks+ 1987	Soda pop 1985	Soda pop 1987
Group 1	636 (21)	727 (24)	253 (8)	316 (10)
Group 2	870(29)	696 (23)*	303 (10)	188 (6)
Group 3	650(21)	622 (21)	134 (4)	161 (5)
All Villages	**762 (25)**	**689 (23)**	**259 (9)**	**219 (7)**

+Soft drinks = sweetened carbonated and non-carbonated beverages

*Significant decrease from 1985 to 1987. p = .001 by analysis of variance

per capita consumption of soda pop in 1985 would be 274 cans, decreasing to 213 cans in 1987. For each data collection period and in all village groups, we found the maximum daily intake of soda pop to be equal to 4 cans per day.

The 1985 consumption pattern for soda pop in the 14 YK Delta villages was less than half the amount reported in Wainwright in 1977. Average per capita intake in that village was reported at 558 cans. This difference could be due to the method of estimating consumption. Measuring food that is imported or purchased does not necessarily reflect actual consumption. We also found large variation between individual villages.

It appears that teenagers in the 14 YK Delta villages are consuming three to four times the amount of soft drinks than teens in the general U.S. population. This may be explained in part by the need for an intake of large amounts of fluids to avoid dehydration due to the consistent low humidity of this region exacerbated by extreme cold during the winter months. Water is not always very appealing because of high sediment and mineral content of some village water supplies. Many households still prefer melted ice for their drinking water.

Tea and coffee are other alternatives that are acceptable to teenagers' taste preferences, but these beverages contain tannic acid, a strong inhibitor of iron absorption. Since iron deficiency is a widespread problem in the Delta, these beverages are not recommended. Fluid milk is not available in most villages, and we found that teens rarely drank milk (made from nonfat, dry powder) outside of school. Finally, artificially sweetened beverages are not available in most village stores. Store managers are reluctant to stock these items because of low or uncertain demand. This leaves the low cost, sweetened powdered mixes such as Koolaid or Tang. As we saw with our consumption figures, this is often the beverage of choice and greatest frequency. These beverages also provided much of the vitamin C in their diets.

Table 2. Percent Change in Daily Per Capita Consumption of Soft Drinks and Soda Pop between 1985 and 1987

Village Group	Soft drinks % Change	Soda pop % Change
Intervention:		
Group 1	+ 14	+ 25
Group 2	- 20	- 38
Group 1 + 2	- 10	- 18
Control:		
Group 3	- 4	+ 20

The percent change in daily per capita consumption of soft drinks and soda pop are presented in Table 2. Despite the difficulties in finding acceptable, nutritionally sound alternatives, we found that when we combined both intervention groups the overall change in soft drink intakes set forth in our objectives was achieved, and these changes were greater in our intervention villages. Although we promoted fruit juices as a nutritious alternative, we found little change in consumption of this beverage. This may have been due to the relative high cost of this food in this region.

Table 3. Mean Daily Consumption of Soft Drinks by Village (grams)

Villages	Soft drinks 1985	Soft drinks 1987	Soda pop 1985	Soda pop 1987
Village Group 1				
Village 1	647	528	190	142
Village 2	591	503	347	230
Village 3	520	1105	186	513
Village 4	778	493	281	240
Village Group 2				
Village 5	286	475	120	158
Village 6	917	1040	362	270
Village 7	582	490	256	211
Village 8	1173	547	234	121
Village 9	1040	949	340	184
Village 10	907	595	329	233
Village Group 3				
Village 11	677	563	137	205
Village 12	450	411	225	51
Village 13	690	897	140	106
Village 14	671	517	85	163

Although intervention activities were going on longer in Group 1 villages as opposed to Group 2 villages, we saw an increase in soft drink and soda pop consumption in Group 1. There was a wide variation in intakes among the four Group 1 villages (see Table 3). Village 3 had an increased consumption of over 100% for both soft drink and soda pop, skewing the average change in consumption for that group. This may have been due to a change in interviewers. There does not appear to be any other economic or availability factors involved. The other three villages more nearly resembled the villages in Group 2.

Table 4. Percent of Subjects Consuming Soft Drinks and Soda Pop

	Percent of Subjects Consuming			
	Soft drinks		Soda pop	
Villages	**1985 (n)**	**1987 (n)**	**1985**	**1987**
Group 1	86 (110)	86 (159)	50	57
Group 2	90 (198)	88 (288)	60	39
Group 3	85 (66)	87 (116)	27	37
Total	**88 (374)**	**87 (563)**	**51**	**44**

n = number of subjects

The data presented in Table 4 represents percent of teenagers consuming soft drinks or soda pop. It is important to note that not all teenagers drank soft drinks or soda pop on the day of the recall. Between 1985 and 1987, the percent of teenagers in our intervention villages consuming soft drinks remained similar with 87% consuming soft drinks in 1985 and 87% in 1987. There was a small increase from 85% to 87% in our control villages. We also see in Table 4 that, contrary to the often heard generalization that teenagers *are* always drinking soda pop, 50% or more of the teenagers we interviewed did not drink soda pop on the day of the interview. The percent of teenagers consuming soda pop decreased from 56% to 45% in the intervention villages and increased from 27% to 37% in the control villages.

SUMMARY

The consumption of sweetened soft drinks by teenagers living in southwestern Alaskan villages is three to four times greater than nationwide estimates. This may be in part explained by limited alternatives to quench thirst and a lack of knowledge of which beverages are healthier. Despite this problem and the general impression that it is difficult to change dietary habits of teenagers, we did see decreases in soft drink intake within our intervention villages that met and exceeded our original objective of a 10% decrease in the amount of soft drinks consumed.

ACKNOWLEDGMENTS

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We want to thank the people of the 14 villages for allowing us to conduct our surveys and for welcoming us into their communities. Recognition should be given to Mary M. Gregory, Anne Dela Rosa, Viola Evan, Felix Lincoln and the staff who helped to collect data and implement our activities. We are grateful for the advice and assistance of Elizabeth Nobmann (Alaska Area Native Health Service, IHS) and Jean Hankin (University of Hawaii, School of Public Health) during all phases of the project.

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Surveillance For Tuberculosis in Alaska, 1986

by Michael Beller, M.D., M.P.H.[1]
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ABSTRACT

The incidence of tuberculosis in the Native population in Alaska (92.2/100,000) continues to be higher than the U.S. tuberculosis incidence (9.3/100,000). This paper describes tuberculosis surveillance in Alaska and estimates the "predictive value positive" for cases recorded by the surveillance system during 1986. (Predictive value positive refers to the proportion of all people identified who actually have the disease.) Active surveillance components included an itinerant chest clinic and survey chest roentgenography program, epidemiologic case investigations, and skin testing. Passive components included cases reported by health-care providers and the U.S. Immigration and Naturalization Service. Half of all new cases were reported by health-care providers. Only 42% of cases were bacteriologically confirmed (vs 87% nationwide) suggesting that some cases may have been false positives. Many cases did not have a contact investigation completed. Recommendations to improve the surveillance system include: 1) following the Centers for Disease Control/American Thoracic Society diagnostic criteria more closely, 2) increasing the number of cases which have contact investigations completed, and 3) targeting surveillance activities towards high risk subgroups such as Southeast Asian immigrants.

INTRODUCTION

During the 1930's and 1940's, published tuberculosis incidence (10,280 per 100,000) and death rates (655 per 100,000) for American Indians and Eskimos living in Alaska were among the highest ever recorded in the medical literature (1,2). In 1947, an evaluation conducted by the American Medical Association concluded that tuberculosis was the most urgent health problem in the Territory of Alaska (3) and although overall tuberculosis incidence in the state has fallen, rates for American Indians and Alaska Natives remain elevated. In 1985, tuberculosis incidence for Alaska's Native population was 92.2 per 100,000 or 10 times the national average of 9.3 per 100,000 (4).

Effective control of an adverse health event requires a comprehensive understanding of the event's epidemiology and ongoing evaluation of its occurrence in the population. Public health surveillance has been defined as "...the collection, collation, and analysis of data and dissemination to those who need to know" (5). Traditionally, surveillance systems have been divided into active and passive activities. An active system routinely and methodically searches for the occurrence of the health event under scrutiny and a passive system relies on reporting by health-care providers. Most surveillance activities in the United States are passive and although all states have statutory provisions requiring providers to report specified conditions, it is well recognized that passive reporting systems generally receive reports on only a portion of cases (6,7). Thus, if complete reporting is important for the investigation or control of an adverse health event, passive reporting usually must be supplemented by active surveillance. Tuberculosis surveillance in Alaska utilizes both active and passive activities.

The goal of the Tuberculosis Control Program, Section of Epidemiology, Alaska Department of Health & Social Services, is the eradication of the disease. Surveillance contributes to this by attempting to identify all persons with tuberculosis disease or infection so that proper treatment or prophylaxis can be provided. The purpose of this paper is to describe the surveillance system for tuberculosis in Alaska during 1986 and to evaluate its "predictive value positive." A companion article in this journal describes the epidemiology of tuberculosis in Alaska (8).

METHODS

Case Definitions:

Surveillance for tuberculosis is concerned with two health events: Tuberculosis disease and tuberculosis infection without disease. Both conditions have had diagnostic standards established by the Centers for Disease Control and the American Thoracic Society (CDC/ATS) (9). According to the CDC/ATS criteria, the definite diagnosis of tuberculosis disease requires bacteriologic confirmation by isolation of *Mycobacterium tuberculosis* from the patient. Ideally, this is accomplished by a positive sputum culture, however, a positive smear also establishes bacteriologic confirmation. In the absence of bacteriologic confirmation, CDC/ATS standards require that three criteria be met: 1) Clinical and/or roentgenographic evidence of current tuberculosis disease, 2) significant reaction to tuberculin skin test, and 3) completion of all diagnostic procedures (9). Patients who meet all three criteria and/or have bacteriologically proven disease can be considered to have tuberculosis. Unless specified otherwise, tuberculosis refers to tuberculosis disease.

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Tuberculosis infection without disease is defined as a reaction of ≥ 10mm induration to 5 tuberculin units of purified protein derivative injected intradermally in a person who does not meet the CDC/ATS criteria for tuberculosis disease. For someone who has been in close contact with a person having tuberculosis, only ≥ 5mm of induration is required to diagnose tuberculosis infection.

Surveillance Activities:

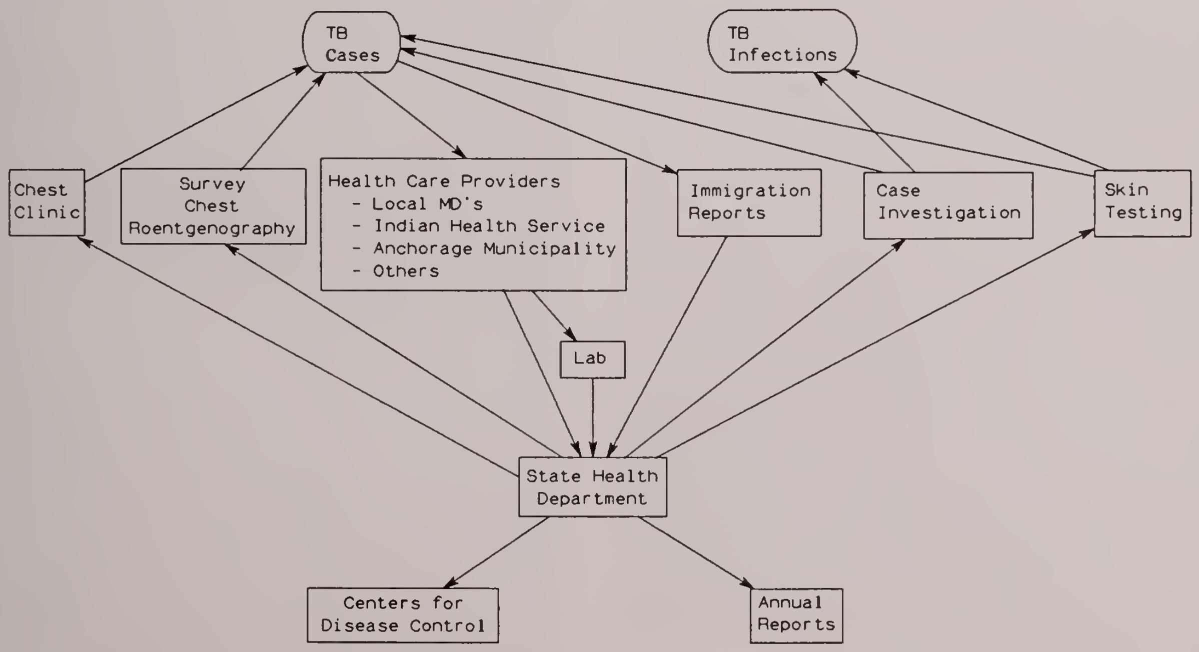
Tuberculosis surveillance during 1986 was conducted by the Section of Communicable Disease Control of the Alaska Department of Health and Social Services.* In 1986, there were six distinct components of the surveillance system (Figure 1). Four activities (itinerant chest clinics, survey chest roentgenography, case investigations, and skin testing) were actively undertaken to detect tuberculosis or tuberculosis infection. These are indicated by the upwards arrows in Figure 1.

Southwestern Alaska (the Yukon–Kuskokwim Delta) has long been an area with especially high rates of tuberculosis (10–12). In 1986, an itinerant x-ray technician employed by the Section took survey chest roentgenograms of 3,343 residents, most were in the Yukon–Kuskokwim Delta. Although many people were examined simply because they were available, a large (but unknown) number of persons were symptomatic and consequently had been referred for roentgenography. The chief of the Section of Communicable Disease Control, a physician board-certified in pulmonary medicine, visited many villages in the Yukon–Kuskokwim Delta region and held clinics to treat people with current disease, examine selected patients with a history of tuberculosis or tuberculosis infection, and follow-up on persons with abnormal roentgenograms.

Skin testing has been an important part of the state's tuberculosis control program since 1959 (13). In 1986, state law required children to be tested at entry or upon transfer to an Alaskan school and at grades K, 1, 3, 7, and 11 (14). All new hospital or nursing home employees were required to have a skin test and most were required to be retested annually.

The final active component was case investigation conducted by either the state Section of Communicable Disease Control or the Anchorage Department of Health and Human Services. Passive reports of tuberculosis (indicated by downward arrows in Figure 1) were received from health-care providers and the U.S. Immigration and Naturalization Service. Since the Section of Communicable Disease Control worked with and encouraged health-care providers to report all tuberculosis cases, this component actually was a "stimulated" passive activity. Surveillance data were compiled

Figure 1. Alaska Tuberculosis Surveillance, 1986



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*In June 1987, reorganization resulted in elimination of the Section of Communicable Disease Control. Responsibility for tuberculosis surveillance and control was assigned to the newly formed Section of Epidemiology, Tuberculosis Control Program.

for quarterly reporting to the CDC, Atlanta, Georgia, as well as for an annual report distributed within the state.

Additional data compiled by the CDC were used to estimate the predictive value positive (PVP) of tuberculosis surveillance in Alaska (15). The methodology used to estimate the PVP of the surveillance system will be explained in the calculations below. Differences between proportions were tested using Chi-square statistics (16).

Table 1. Sources of Reported Tuberculosis Cases, Alaska, 1986

Active Surveillance	Incident Cases	Persons Evaluated
Chest Clinic	2	937
Survey Chest Roentgenograms	6	3,343
Case Investigations	4	34
Skin Testing	12	90,684
Passive Surveillance		
Immigration Reports	7	—
Reports from Providers	36	—
Unknown	5	
	72	

RESULTS

During 1986, 72 new cases of tuberculosis were reported to the Section of Communicable Disease Control. In Table 1, each is classified according to the component of surveillance system which identified the person. For each active component, the number of skin tests, case investigations, roentgenograms, etc. is also shown.

Thirty-six (50%) of the incident cases were reported by health-care providers. Of these, 14 were reported by the Indian Health Service (IHS), 11 by local physicians, and the remainder by other medical providers. Survey roentgenograms detected six cases of tuberculosis. There were 937 patient visits and two new diagnoses of tuberculosis in the chest clinics held by the Section of Communicable Disease Control. Twelve persons with tuberculosis (11 of which were from the school testing program) and 1,522 persons with infection were discovered by the 90,684 skin tests.

Twenty (67%) of 30 bacteriologically confirmed cases and 14 (33%) of 42 cases without bacteriologic confirmation had investigations of household members and close personal contacts completed. These 34 contact investigations were conducted by either the Anchorage Department of Health and Human Services or the Section of Communicable Disease Control. As a result, four persons with tuberculosis (all from investigations of bacteriologically confirmed cases) and 18 persons with tuberculosis infection were identified.

Predictive Value Positive:

Predictive value positive (PVP) is a measure frequently used by clinicians to help interpret the results of a screening test. If a patient with an undiagnosed condition has a positive test (for example, an elevated erythrocyte sedimentation rate), the physician needs to have some idea of the probability that the patient has a certain disease (for example, rheumatoid arthritis). Specifically, the PVP of a test is defined as the percentage of all people with a positive result (an elevated ESR in this example) who actually have the disease (rheumatoid arthritis in this example). This can be stated mathematically as:

$$\text{PVP} = \frac{\text{true positives}}{\text{all positives}}$$

For the example given, if out of 100 people with an elevated ESR, 33 are subsequently confirmed to have rheumatoid arthritis, then the PVP of an elevated ESR is 33% for rheumatoid arthritis.

The PVP of a surveillance system is an important measure of the system's effectiveness. In the ideal situation, the PVP is 100% — that is, all cases in the system have the condition or disease under study. On the other hand, if a surveillance system detects people who actually do not have the disease, then the PVP will be less than 100%. In this case, many "cases" have been misdiagnosed, counted by the surveillance system, and treated for a disease that they do not have.

In order to estimate the PVP of the Alaska tuberculosis surveillance system, it is necessary to know how many of the 72 cases actually had tuberculosis. Thirty cases were bacteriologically proven and therefore must have had the disease. The remaining 42 cases (58%) lacked bacteriologic confirmation, but were counted by the tuberculosis surveillance system because they were considered to have tuberculosis by the chief of the Section of Communicable Disease Control. Whether or not these persons met the CDC/ATS criteria required to diagnose tuberculosis in the absence of bacteriologic confirmation cannot be determined. In sharp contrast, only 13% of nationwide (excluding Alaska) tuberculosis cases reported to the CDC for 1986 lacked bacteriologic confirmation (15). The difference between the Alaska proportion without bacteriologic confirmation (58%) and the corresponding proportion for the U.S. (13%) is highly statistically significant (Chi-square = 125.6; 1 d.f.; $p<0.000001$) and raises doubt about the inclusion of so many bacteriologic negative cases in Alaska.

If it is assumed that all non-Alaska U.S. cases actually had disease, (i.e., no false positives reported to the CDC) and that the proportion of Alaska cases with bacteriologic confirmation should closely follow the national proportion, then it is possible to calculate the predictive value of a "positive result" (i.e., being recorded as a tuberculosis case) in the Alaska tuberculosis surveillance system. In the U.S. (less Alaska), of 22,692 tuberculosis (TB) cases in 1986, 19,745 were bacteriologically confirmed (BC) while 2,947 were either bacteriologically negative or bacteriologic status unknown (15). This allows calculation of the following ratio:

$$\frac{\text{TB cases with BC}}{\text{TB cases without BC}} = \frac{19{,}745}{2{,}947} = \mathbf{6.70}$$

This means that, in the U.S., on average, there were 6.70 bacteriologically confirmed cases for each tuberculosis case without bacteriologic confirmation.

In Alaska during 1986, 30 tuberculosis cases were bacteriologically confirmed. For the 6.70:1 ratio to hold, the number of Alaska cases without bacteriolgic confirmation would have to have been four.

In a tuberculosis surveillance system, the number of true positives is the sum of the number of cases with bacteriologic confirmation plus the number of cases meeting CDC/ATS criteria but without bacteriologic confirmation. Thus, true positives in Alaska can be estimated as 30 + 4 = 34. Therefore;

$$\mathbf{PVP} = \frac{\text{true positives}}{\text{all positives}} = \frac{34}{72} = \mathbf{.47}$$

This indicates that over half of the people counted by the tuberculosis surveillance system as having disease may have been falsely designated (and treated) as having tuberculosis.

DISCUSSION

In view of the large differences between Alaska and national rates of bacteriologic confirmation, the low PVP is not surprising. However, there are several limitations to this analysis. First, since rates of bacteriologic confirmation are lowest for younger persons, age can confound the relationship between tuberculosis and sputum positivity. However, the age distribution of Alaska cases is similar to that of U.S. cases (8) so age would not be expected to play an important role in the low rate of bacteriologic confirmation in Alaska. Secondly, it was assumed that all non-Alaska U.S. cases were true positives. Possibly some of the U.S. cases without bacteriologic confirmation did not actually have tuberculosis and, therefore, were false positives. If an adjustment were made for the inclusion of false positives in the U.S. data, the PVP for Alaska would be even smaller. Thirdly, the approach used for estimating PVP ignores the unique challenges intrinsic to diagnosing tuberculosis in rural Alaska (such as the distance from a laboratory and the absence of bronchoscopy) and assumes that the proportion of bacteriologically positive cases in the state should closely follow the nationwide trend. Essentially, this assumption ignores differences between Alaska and the rest of the United States and assumes that uniform diagnostic criteria should be applied. Finally, it is conceivable that the low proportion of bacteriologically confirmed cases in Alaska could result from earlier diagnosis of persons with tuberculosis in Alaska than persons with tuberculosis elsewhere. However, half the Alaska tuberculosis cases were passively reported by health-care providers and there is nothing to suggest that these patients seek medical care or are diagnosed earlier than patients elsewhere. Furthermore, early diagnosis of persons who are bacteriologically negative is unlikely since radiographic and clinical changes suggesting the disease will not have had time to occur. Thus, the low PVP of tuberculosis surveillance in Alaska in 1986 appears to be real.

Tuberculin skin test results were available for over 90,000 persons, roughly one-sixth of the state's population during 1986. Skin test conversion is a "sentinel health event" and each convertor requires careful investigation to identify possible contact with a person with infectious tuberculosis. However, only 1.7% of the skin tests were positive. The skin testing program should be carefully evaluated to help determine high-risk areas, evaluate current programs and guide future activities. The need to test such a large portion of the population should be reassessed.

There is a high incidence of tuberculosis in certain subpopulations in the U.S.. For example, tuberculosis rates are higher for the homeless, persons with HIV infection, and Southeast Asian refugees and immigrants. In Alaska during 1986, there were no systematic surveillance activities in these populations. The tuberculosis control program should assess the prevalence of tuberculosis infection in these groups and, if found to be high, provide for specific surveillance and control measures.

In mid-1987 the CDC reviewed the Alaska Tuberculosis Control Program and issued a report which is consistent with the findings presented here (17). The CDC review as well as the National Center for Devices and Radiological Health of the U.S. Food and Drug Administration (18) have stated that routine chest roentgenography in unselected populations is unjustified, and accordingly routine screening roentgenography was stopped in 1987.

CONCLUSIONS

Three major conclusions can be drawn from this evaluation. First, CDC/ATS diagnostic criteria should be followed more carefully. The proportion of cases with bacteriologic confirmation in Alaska fell far short of those reported by the rest of the country and the PVP of the Alaska surveillance system was low. Second, contact investigations need to be undertaken more consistently. Unlike surveillance for other infectious diseases, tuberculosis surveillance attempts as close to 100% case ascertainment as possible and contact investigations and other active surveillance activities will continue to play an important role in tuberculosis control. Finally, surveillance needs to be directed towards high risk subgroups such as the homeless, persons with HIV infection, and Southeast Asian refugees.

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The Epidemiology of Tuberculosis in Alaska, 1987

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ABSTRACT

Alaska's tuberculosis morbidity and mortality rates have declined dramatically since the 1950's. At the same time, the epidemiologic characteristics of the disease in Alaska have changed. The tuberculosis incidence rate is now highest among persons of Asian ancestry, particularly the foreign-born. The high rate of disease in Alaskan children indicates that active transmission of *Mycobacterium tuberculosis* to previously uninfected persons is still occurring. The large proportion of cases among persons under the age of 35 implies a failure in the detection of tuberculosis infection and/or in the use of isoniazid chemoprophylaxis. Alaska's tuberculosis control efforts must focus on ensuring that persons with tuberculosis disease are appropriately and fully treated, on detecting tuberculosis infection in persons eligible for isoniazid preventive therapy, and on applying aggressively the preventive therapy recommendations of the American Thoracic Society and the Centers for Disease Control.

INTRODUCTION

Historically, tuberculosis in Alaska predominated among Alaska Natives (Aleuts, Eskimos, and Indians). The mean annual death rate among Alaska Natives from tuberculosis during the years 1926–1930 was 655 per 100,000 (1), and tuberculosis-attributable death rates among Alaska Natives during the second quarter of this century "were among the highest ever recorded for the disease" (2). As recently as 1952, the incidence rate of tuberculosis among Alaska Natives was 1,854 cases per 100,000 (2), and the death rate from tuberculosis was 501 deaths per 100,000 (3).

Intensive case finding using statewide x-ray screening surveys, a short-lived BCG vaccination program, and intensive direct supervision of treatment of persons with tuberculosis infection or disease following the introduction of chemotherapy in the early 1950's resulted in a rapid decline in tuberculosis morbidity and mortality rates and in rates of infection in children.

Extensive tuberculin skin-testing conducted from 1948–1951 as part of the Alaska Department of Health's BCG vaccination program showed that 89% of a sample of 360 Eskimo children 5–8 years of age living in the Yukon–Kuskokwim Delta were tuberculin reactors (4). The same survey demonstrated lower rates of tuberculin reactivity among Northwest Alaska Eskimo children (67%) and Interior Alaska Indian children (65%) of the same age group; in contrast, only 0.6% of 363 white children from Southeast Alaska were found to be tuberculin reactors.

A series of tuberculin skin-test surveys of Yukon–Kuskokwim Eskimo children three years of age or younger, conducted during the period of the most intensive tuberculosis control efforts, demonstrated a rapid decline in tuberculin reactivity among successive birth cohorts (5). Of the first sample of children under age four tested during 1949–1951, 34.4% were tuberculin reactors. By 1957, only 14.4% of children in this age group were tuberculin-positive; and in 1960 the infection rate in children born since the previous survey had fallen to 1.7%.

By 1970, the rate of new active cases of tuberculosis among Alaska Natives has fallen to 154 per 100,000. It dropped further to 79.0 cases per 100,000 in 1985 (Table 1).

Moreover, the number of newly identified tuberculin reactors has declined to low levels. Of 71,316 Alaskan children of school-age and younger who were not known to be tuberculin-positive and who were tuberculin skin-tested in 1986, only 152 (0.21%) were tuberculin reactors (6). In the same year only 937 (4.83%) of 18,431 adults not previously known to be tuberculin reactors were found to be tuberculin-positive (6).

The massive public health interventions begun in the 1940's and 1950's have dramatically changed the epidemiologic characteristics of tuberculosis in Alaska. This paper will describe the epidemiology of tuberculosis in Alaska during recent years.

Tuberculosis Case Definition

For the purposes of case-counting and reporting, the American Thoracic Society and the Centers for Disease Control (7) define a case of tuberculosis as an individual from whom a sample of tissue or body fluid has yielded *Mycobacterium tuberculosis* on culture. In the absence of bacteriologic confirmation, an individual may be counted as a case of tuberculosis if diagnostic evaluation has been completed and if the following other criteria have been fulfilled: (a) the individual has (or has had) a significant tuberculin skin test reaction or has previously had culture-confirmed tuberculosis disease; (b) there are signs and/or symptoms compatible with tuberculo-

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Table 1. Tuberculosis Cases and Incidence Rates* in Alaska, by Race**, 1981-1987

	WHITE		BLACK		NATIVE		ASIAN	
YEAR	No.	(RATE)	No.	(RATE)	No.	(RATE)	No.	(RATE)
1981	15	(4.5)	1	(6.8)	56	(80.7)	11	(90.6)
1982	20	(5.6)	4	(25.5)	60	(81.6)	12	(93.3)
1983	23	(6.0)	1	(5.9)	62	(78.5)	12	(86.8)
1984	19	(4.7)	0	(0.0)	46	(55.1)	14	(95.9)
1985	22	(5.3)	1	(5.5)	68	(79.0)	19	(126.2)
1986	12	(2.8)	2	(10.7)	35	(40.1)	23	(150.5)
1987	11	(2.7)	3	(16.4)	31	(36.1)	16	(106.6)

***Rate per 100,000 population.**

****Population estimates for each racial group are based on the assumption that the proportion of the population attributable to each group has remained constant since the 1980 census. Annual state-wide population estimates used for these calculations were supplied by the Alaska Department of Labor.**

sis, such as clinical evidence of current disease or an abnormal, unstable (worsening or improving) chest x-ray; and (c) a decision has been made to treat the individual with two or more anti-tuberculosis medications.

Reported Cases and Incidence Rates

Figures 1 and 2 depict the tuberculosis incidence rates per 100,000 population reported annually by the Alaska Department of Health & Social Services during the past thirty-six years (1952-1987). Clearly, there has been a steady downward trend in tuberculosis incidence rates during the period. The incidence rate for 1987, 11.3 new active cases per 100,000 residents, was less than half the rate for 1978. Nevertheless, Alaska's rates during 1985 and 1986 were higher than the tuberculosis incidence rates for the entire United States, which were 9.3 and 9.4 per 100,000, respectively (8). The 110 new cases of tuberculosis reported in Alaska in 1985 yielded a rate of 20.4 cases per 100,000, the highest among the 50 states for that year (9). During the following year there were 72 cases reported, a rate of 13.2 per 100,000, high enough to rank Alaska eighth among the states (9).

Only about half of the tuberculosis cases diagnosed in Alaska during 1985-1987 were confirmed by culture (Table 2), but the incidence rate of bacteriologically confirmed cases—as high as 8.7 per 100,000 in 1985—is substantial when compared to overall national rates during the same period.

Only five (9.3%) of the 54 cases of tuberculosis diagnosed in children 0-14 years of age during 1985-87 were confirmed by culture, compared to 111 (59%) of 189 cases in persons older that 14 years of age. Seven of the eight patients in the under 15-year age group reported during 1987 were younger than eight years; of these seven, only one was cultured. In 1987,75% of cases older than 14 years were culture-confirmed, compared with 54% and 49% in 1985 and 1986, respectively.

Distribution By Age and Sex

Sixty-one cases of tuberculosis were diagnosed among Alaska residents in 1987 (Figure 3). Their ages ranged from 10 months to 82 years (mean, 42.1 years; median, 41 years). Eleven (18%) of the cases were less than 20 years of age. Five cases were younger than 5 years, and three were aged 5-9 years. The finding of tuberculosis in children, in whom disease is not the

Table 2. Bacteriologically confirmed cases of tuberculosis, by age group, 1985-1987

	0-14 YEARS			15+ YEARS		
YEAR	No. of Confirmed Cases	Total No. of Cases	% of all Cases in Age Group	No. of Confirmed Cases	Total No. of Cases	% of all Cases in Age Group
1985	2	27	7%	45	83	54%
1986	3	19	16%	26	53	49%
1987	0	8	0%	40	53	75%
Total	5	54	9%	111	189	59%

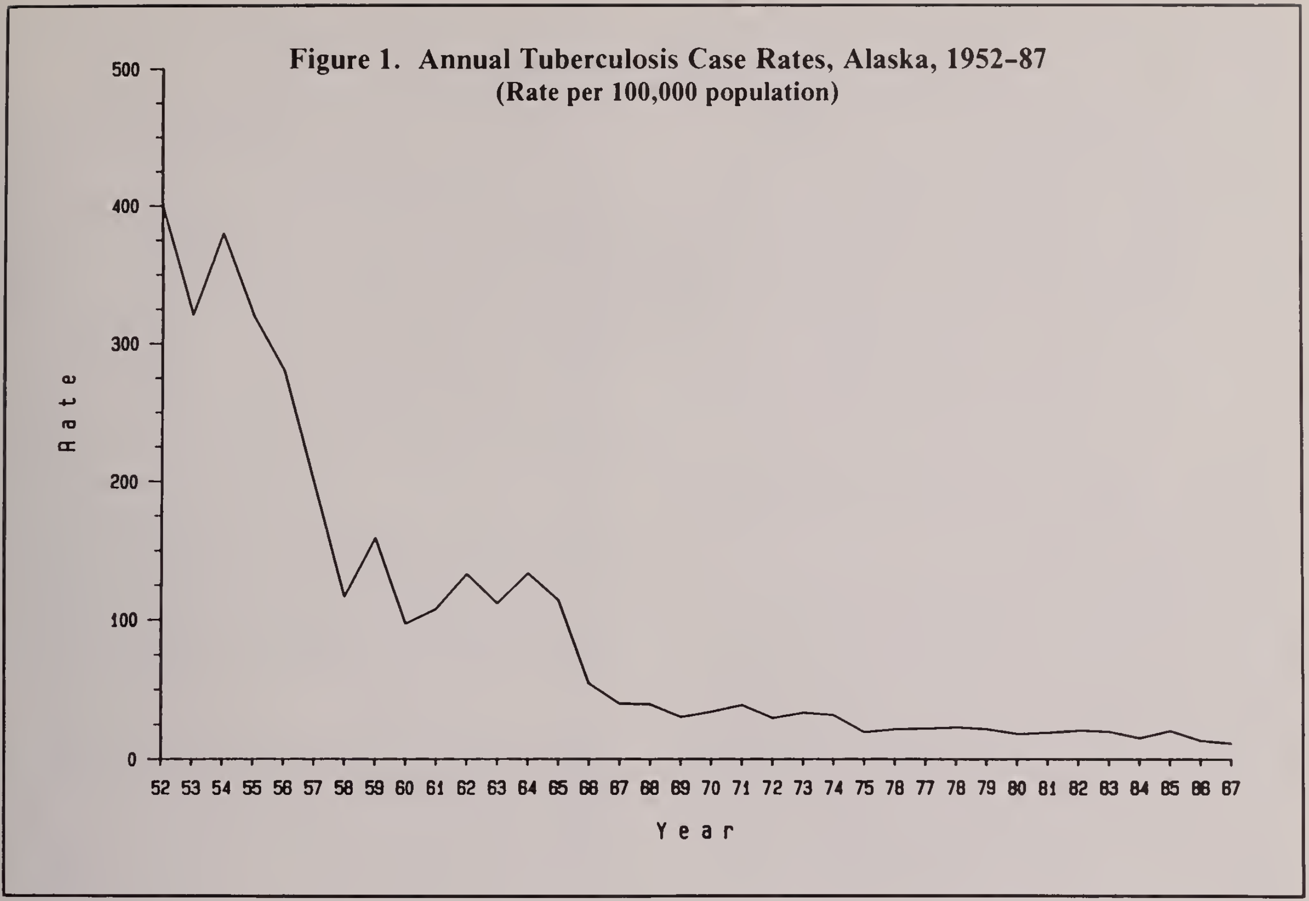

Figure 1. Annual Tuberculosis Case Rates, Alaska, 1952–87
(Rate per 100,000 population)

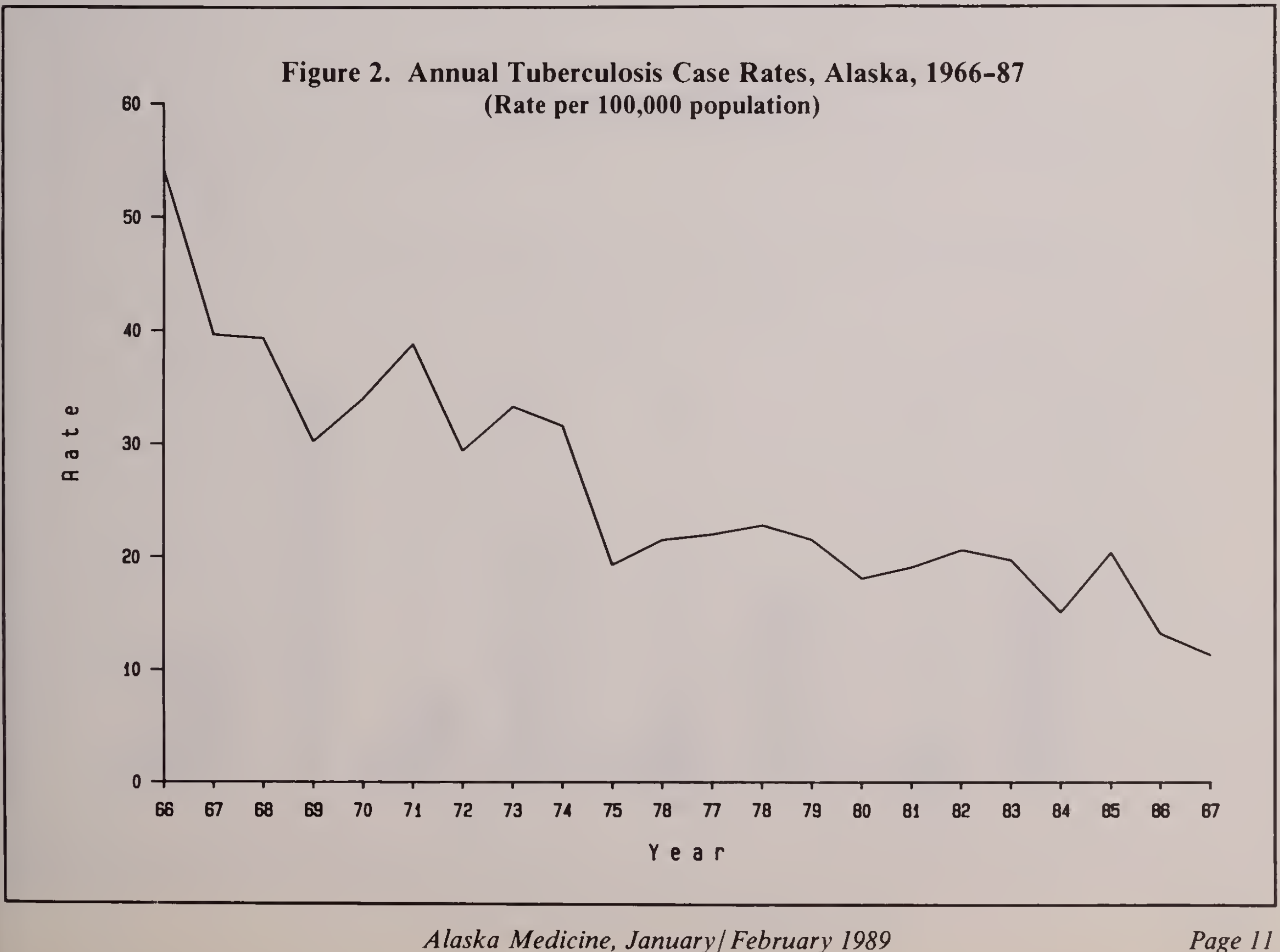

Figure 2. Annual Tuberculosis Case Rates, Alaska, 1966–87
(Rate per 100,000 population)

result of activation of "old" infection, is proof of active transmission of tuberculosis from adults with infectious pulmonary tuberculosis to uninfected children.

The age-group distribution of cases is different for each racial/ethnic group (Figure 3). Only 3 cases occurred among blacks: the three oldest age groups had one case each. For whites, there was a direct correlation between numbers of cases and increasing age; 10% of white cases were in the 0–19 age group, whereas 40% occurred in persons older than 49 years. Of the 14 Asian cases, half were in persons under 35 years of age; six (43%) were in the oldest age category. In contrast, the majority of cases among Alaska Natives were among the middle-aged and elderly; sixty-two percent were 35 years of age or older—evidence of the persisting effect of high rates of tuberculosis infection and disease during the past several decades.

During 1985 and 1986 greater numbers of cases were diagnosed among younger age groups, whereas in 1987 cases were more evenly distributed. It is noteworthy that cases within the under 35-year age group accounted for 122 (50.2%) of the 243 tuberculosis cases reported during 1985–1987 (Table 3). These are cases which should be considered potentially preventable, since the American Thoracic Society and the Centers for Disease Control currently recommend that all tuberculin reactors under the age of 35 years be offered isoniazid chemoprophylaxis (10).

The greatest and most consistent differences between Alaska and U.S. rates in 1985 and 1986 are in the 5- to 14-year age range and in the 55-year-plus age group (Table 4). There were substantial differences in Alaska's age-group incidence rates for the two years, as might be expected in a relatively small population; the largest differences are in the 0-4, 35-44, 45-54, and 65+ age groups. The U.S. rates, on the other hand, show much more year-to-year consistency. The U.S. incidence rate declines from ages 0–4 years to 10–14 years, then gradually increases with age to a maximum among persons over 64 years of age. Alaska's trend is similar except for high rates in the 5- to 14-year age range, which has the lowest rates among the U.S. population. The U.S. rate for the 5–14 age group was 1.4 per 100,000 for both 1985 and 1986. Alaska's age-adjusted rates for that age group for those years were 25.6 and 19.4, respectively—rates 14–18 times as high as U.S. rates. Thus, both Alaska and the U.S. have high incidence rates among the elderly (though Alaska's rates are higher), and Alaska's rates in the 5–14 age category substantially exceed the low U.S. rates in that age category.

During the past three years (1985–1987), 153 (63%) of the 243 reported tuberculosis cases were men. Women accounted for only 37% of the cases. This is nearly identical to the sex distribution of tuberculosis cases reported for the United States during 1985 and 1986: 65% of all U.S. cases were males; 35% were females (8).

Figure 3. Cases of Tuberculosis in Alaska, 1987
By Race within Age Group
N = 61

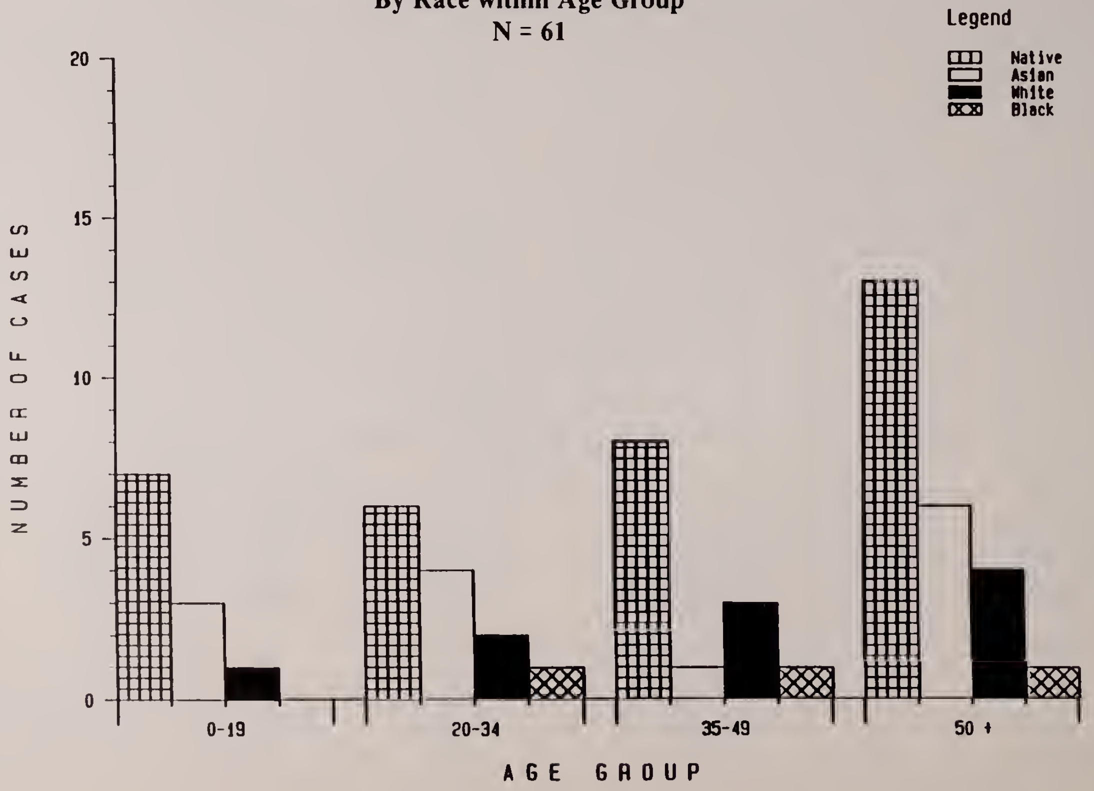

Table 3. Tuberculosis cases in Alaska, by age group, 1985–1987

AGE GROUP (YRS.)	1985		1986		1987	
	No.	(%)	No.	(%)	No.	(%)
0–19	34	(31)	24	(33)	11	(18)
20–34	23	(21)	17	(24)	13	(21)
35–49	21	(19)	9	(13)	13	(21)
50–64	18	(16)	16	(22)	12	(20)
65+	14	(13)	6	(8)	12	(20)
TOTAL	110	(100)	72	(100)	61	(100)

Distribution By Race

The incidence rate of tuberculosis among Alaska's whites is consistently low, varying from 6.0 cases per 100,000 in 1983 to 2.7 cases per 100,000 in 1987 (Table 1). For comparison, incidence rates for whites (Hispanic and non–Hispanic combined) in the United States as a whole during 1985 and 1986 were 5.6 and 5.7 cases per 100,000, respectively (8).

Rates for blacks are somewhat higher than for whites, but the numbers of cases among blacks have been consistently low. Excluding 1984 (during which no cases among blacks were recorded), incidence rates have ranged from 5.5 to 25.5 per 100,000, compared with national rates of 26.7 and 27.6 per 100,000 in 1985 and 1986, respectively (3).

During 1985–1987, members of ethnic/racial minorities comprised 81.5% of all cases of tuberculosis reportedin Alaska. The tuberculosis incidence rate has in the past been highest among Alaska Natives. However, during the period 1981–1987, the incidence rate was highest among Asians during each of the seven years. While the rate among Natives declined from 80.7 cases per 100,000 at the beginning of the period to 36.1 per 100,000 in 1987, the incidence rate among Asians increased from 90.6 cases per 100,000 in 1981 to rates well in excess of 100 cases per 100,000 during 1985–1987. These rates are more than twice the incidence rates reported for Asians and Pacific Islanders in the United States during 1985 and 1986 (8). Although the incidence rate of bacteriologically confirmed disease among Asians in Alaska during 1987 was only 60.0 cases per 100,000 (9 culture–confirmed cases among an estimated population of 15,005 Asians and Pacific Islanders), this rate exceeded the case rates cited for this group in the United States for 1985 and 1986 (8).

Fifteen (25%) of Alaska's tuberculosis cases reported in 1987 were immigrants. The diagnosis of tuberculosis was made after their arrival in the United States in all but one person. Of these 15 individuals, one was from the Dominican Republic and fourteen were from Asian

Table 4. Tuberculosis incidence rates in Alaska and in the United States, by age group, 1985–1986

Age Group (Yrs.)	1985		1986	
	Alaska	U.S.A.	Alaska	U.S.A.
0–4	9.9	4.4	4.9	4.0
5–9	26.1	1.6	21.5	1.6
10–14	25.1	1.2	17.3	1.3
15–19	15.6	2.5	10.2	2.8
20–24	13.3	5.8	9.4	5.9
25–34	12.6	8.5	9.3	9.2
35–44	16.2	10.1	8.0	10.2
45–54	32.0	12.8	15.8	13.1
55–64	39.5	14.6	38.9	14.0
65+	77.2	22.3	32.6	21.9
Total	27.1*	9.3	16.5*	9.4

***Rate age–adjusted to same–year U.S. population.**

countries (Philippines, 6; Korea, 5; China, 2; and India, one). All of the immigrant cases lived in major cities or towns: nine (60%) in Anchorage; three (20%) in Fairbanks; and one each in Kodiak, Juneau, and Ketchikan.

Geographic Distribution

Thirty-two (52%) of the 61 cases reported in 1987 lived in a total of eight communities with populations greater than 2,500 persons; twenty-nine (48%) were residents of sixteen villages, all with populations of fewer than 1,000. Seventeen (28%) were Anchorage residents, and six (10%) resided in Fairbanks. Twenty-seven other cases were distributed among 21 communities, with no more than two cases in any one community (Figure 4).

The remaining eleven cases, however, all occurred in a rural western Alaskan Village with a population of approximately 250. The investigation of this outbreak led to the screening (tuberculin skin-testing and/or chest x-rays) of 227 persons in three villages. Besides the 11 active tuberculosis cases, 13 newly infected individuals were discovered. The index case was a 24-year old woman with cavitary pulmonary tuberculosis. Three cases, including the index case, required quarantine and hospitalization for medical treatment under direct supervision for eight weeks. Exclusive of hospitalization expenses for these three patients, the estimated total cost of investigating this outbreak, of medically evaluating infected persons, and of treating those with tuberculosis infection or disease was $21,000.

Site of Disease

Of the 61 cases of tuberculosis diagnosed during 1987, 50 (82%) had pulmonary disease. Five of these 50 cases also had disease in at least one additional site: two had pleural involvement; one, lymphatic disease; one, genitourinary; and one, disseminated disease.

Eleven cases (18%) had extrapulmonary disease only. Six had cervical and/or intrathoracic lymphatic involvement; two had genitourinary disease; two had pleural tuberculosis; and one had peritoneal involvement only.

One patient—a person with extrapulmonary disease confined to the intrathoracic lymph nodes—was the first individual in Alaska reported to have both tuberculosis and AIDS.

Drug-Resistant Tuberculosis

In Alaska's public health laboratories, routine antibiotic susceptibility testing is performed with fixed concentrations of isoniazid, rifampin, and streptomycin. Eight (.73%) of all tuberculosis cases reported in Alaska in 1985 were associated with organisms resistant to anti-tuberculosis drugs, and four (5.6%) of all cases in 1986 were caused by such organisms. In all instances, organisms were resistant to only one of the three antibiotics against which they were tested. Mycobacteria from 9 of the 12 cases showed varying degrees of resistance to rifampin; those from the other 3 patients were resistant to streptomycin. There were no instances of isoniazid resistance. Eight of the 12 patients with drug-resistant organisms were Alaska Natives; two were of Asian descent; and two were Caucasian. There was no record of any of these patients' having been previously treated with the antibiotic to which their tubercle bacilli were resistant. Of the nine patients with rifampin-resistant organisms, two were treated with regimens of three or more drugs. The other seven patients, treated with a combination of isoniazid and rifampin despite known resistance to rifampin, had radiographic improvement and/or conversion of mycobacterial cultures to negative.

There were no known instances of drug-resistance among cases diagnosed in 1987. No information regarding antibiotic susceptibility was available for two patients with culture-confirmed tuberculosis.

Recurrent Tuberculosis

Eight (13%) of the 1987 cases had at least one prior diagnosis of tuberculosis. The time intervals since each patient's most recent previous diagnosis of tuberculosis ranged from 8 to 33 years (mean, 27.5 years; median, 31.0 years). All of these patients were more than 35 years old. In addition, all had been treated with some anti-tuberculosis chemotherapy for their prior episode of tuberculosis. Five patients had taken regimens of two or more drugs (isoniazid/PAS, 4 cases; isoniazid/rifampin/ethambutol, one case). The regimens of three patients were not specified. Details regarding the exact duration of, or patients' compliance with, medical therapy were not recorded.

DISCUSSION

(1) Though the greatest number of tuberculosis cases in Alaska are Alaska Natives, the highest race-specific incidence rate in Alaska is among Asians, especially among the foreign-born. Fourteen (88%) of the 16 Asian tuberculosis cases reported in Alaska in 1987 were foreign-born. Tuberculosis is relatively uncommon among whites and blacks in Alaska. It is reasonable, then, to concentrate Alaska's tuberculosis control efforts among its Native and Asian populations.

(2) Alaska's older citizens (those 55 years of age and over) have high rates of tuberculosis, as do the elderly in the general U.S. population. However, Alaskans aged 5-14 years have much higher rates of disease than their counterparts in the general U.S. population. High disease rates in children indicate active transmission of tuberculosis to previously uninfected children from adults with pulmonary tuberculosis.

(3) Since 39%-56% of cases reported during 1985-1987 occurred among persons under age 35, and since isoniazid chemoprophylaxis is recommended for all tu-

berculin reactors in that age group, appropriate screening and prescription of preventive therapy for all tuberculosis-infected persons under age 35 will result in a substantial reduction in the number of tuberculosis cases occurring annually in Alaska.

(4) Recurrent tuberculosis is not uncommon in Alaska. The majority of persons reported to have recurrent tuberculosis in 1987 had been treated previously with chemotherapeutic regimens less effective than those now available. The anti-tuberculosis therapeutic regimens currently recommended by the American Thoracic Society and the Centers for Disease Control are associated with relapse rates of under 5% (11,12). However, in order for these regimens to achieve this level of effectiveness, they must be appropriately prescribed, and efforts must be made to ensure patients' compliance with prescribed medical therapy.

(5) Focal epidemics of tuberculosis still occur in Alaska. It is important to conduct epidemiologic investigations of close contacts of all cases of pulmonary tuberculosis (in order to identify additional cases and infected contacts) and to investigate intensively the associates of young children with tuberculosis infection (in order to identify the source of infection).

(6) Drug resistant tuberculosis is not a major problem in Alaska. Resistance to a single drug—most often rifampin—is the most common pattern.

CONCLUSION

Efforts to control the epidemic of tuberculosis in Alaska that ravaged Alaskan Natives through the 1950's achieved near-miraculous results. Now, by applying new control strategies based upon careful epidemiologic analysis of tuberculosis, further major reductions in morbidity and mortality can be expected. Indeed, there are realistic hopes that one day tuberculosis can be eradicated.

The Division of Public Health is committed to implementing the "Strategic Plan for the Elimination of Tuberculosis" developed by the national Centers for Disease Control (13,14). Key elements are presented in an accompanying article in this issue of *Alaska Medicine.*

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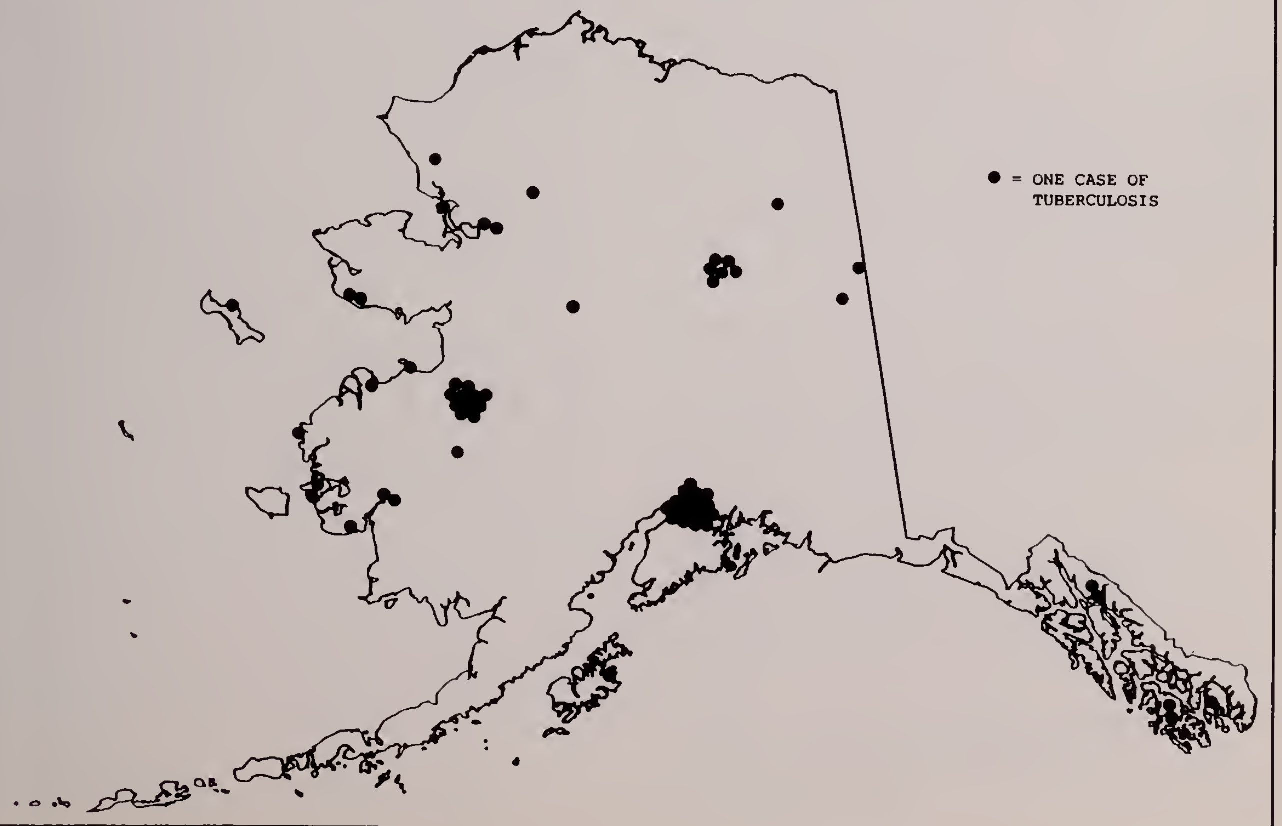
Figure 4. Distribution of Tuberculosis Cases, by Place of Residence, 1987



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Strategy for the Elimination of Tuberculosis in Alaska

Section of Epidemiology
Division of Public Health
January, 1988

INTRODUCTION

In June 1987, epidemiologists from the Division of Tuberculosis Control, Centers for Disease Control (CDC), conducted a review of the Alaska State Tuberculosis Control Program and submitted their observations and recommendations to the Section of Epidemiology. Section of Epidemiology and CDC reviewers consulted extensively with individuals and agencies involved in controlling tuberculosis. We are delighted with the widespread support that has been expressed for implementing program changes to improve our efforts to control tuberculosis in Alaska. We thank all those who contributed their valuable time and wisdom.

THE GOAL OF THE ALASKA DIVISION OF PUBLIC HEALTH IS TO ELIMINATE TUBERCULOSIS IN ALASKA.

Strategies for the Elimination of Tuberculosis

1. ***Diagnosis and Treatment of all Culture-Confirmed Cases of Pulmonary Tuberculosis.*** These are the patients most likely to transmit infection. Prompt identification and appropriate treatment of these cases are essential to limiting the spread of tuberculosis.

2. ***Epidemiologic Investigation of Close Contacts of all Cases of Pulmonary Tuberculosis.*** Infected contacts should receive appropriate chemotherapy in order to prevent future tuberculosis disease.

3. ***Effective Skin-Test Screening Programs*** to identify (and to treat, when appropriate) other individuals infected with *M. tuberculosis.*

   Periodic Skin-Testing of School Children.

   - Routine skin-testing by grades. (Skin-testing of students in pre-elementary school and in grades K,1,3,7, and 11 is required by state law.)
   - More extensive testing in schools with a large Native/Asian population or with a significant proportion of tuberculin reactors among the student population.
   - Preventive therapy for all infected children.
   - Screening of close contacts of newly infected children in order to identify the source of infection and other infected associates.

   Screening of High-Risk Adult Populations. (For example, Asians, Natives, the homeless.)

4. ***Assistance to Insure Meticulous Compliance with Drug Treatment.***

These efforts should reduce the transmission of *M. tuberculosis* and shrink the population of infected persons at risk of developing tuberculosis disease. Any actions on the part of patients, health care providers, or public health personnel which result in the failure of these strategies will impede the eradication of tuberculosis from our state.

Components of Alaska's Tuberculosis Control Program

1. ***Tuberculosis Case Register***

   The case register contains data on patients with active or inactive tuberculosis disease and on investigated contacts of those cases. It enables public health personnel to keep track of diagnostic information on each patient with tuberculosis, their clinical management, and their compliance with recommended therapy.

2. ***Provision of Anti-Tuberculosis Drugs***

   The Section of Epidemiology supplies anti-tuberculosis chemotherapy **free of charge**, for the duration of treatment, to patients being treated for tuberculosis infection or disease.

3. ***Laboratory Services***

   The Section of Laboratories provides, **free of charge** to the patient, level 1 and 2 laboratory services, including AFB smears, mycobacterial cultures and antibiotic susceptibility testing of mycobacterial isolates.

4. ***Expert Consultation is available to physicians and all other health care providers regarding:***

   a. Clinical Diagnosis

b. Treatment and management of tuberculosis disease
c. Preventive therapy

5. *Disease Reporting and Surveillance*

The goal of surveillance is the detection of all persons with tuberculosis infection or disease, for the following purposes:

a. Treatment and control of disease.
b. Program planning and evaluation:
   - Data analysis—determination of incidence rates by age groups, sex, race, geographic area, and subpopulation (e.g., school children).
   - Identification of high-risk groups.
   - Provision of outreach programs to persons at increased risk of developing tuberculosis disease/infection (for example, the Asian community, the urban homeless).
   - Formulation of State Tuberculosis Control policies.
   - Evaluation of effectiveness of program activiies.

IMPORTANT CHANGES IN PROGRAM POLICIES/RECOMMENDATIONS

1. *Patient Care*

The Tuberculosis Control Program will no longer be involved in direct clinical patient care or management. Evaluation and treatment of patients with suspected or documented tuberculosis disease or infection should be conducted by a primary care provider of each patient's choice. The Tuberculosis Control Program will always assist in any circumstance to see that all patients have a "medical home," where their clinical needs can be optimally provided for.

2. *Elimination of the Use of Chest X-Rays for Screening*

Chest x-ray examinations of unselected populations are not sufficiently productive as a screening device for tuberculosis, and their routine use for such purpose cannot be recommended. Chest x-ray examinations should be obtained only when a specific medical indication exists (e.g., relevant history, symptoms, and/or significant tuberculin skin test reaction).

The use of chest x-rays for pre-employment screening, for routine follow-up of tuberculosis patients who have completed treatment, as a periodic screening tool in long-term care facilities, or as a periodic evaluation tool for tuberculin reactors should be discontinued.

As an indicator of response to tuberculosis treatment, chest x-ray examinations are less reliable than results of sputum smear/culture and assessments of symptoms and clinical status; thus, routine periodic chest x-ray examinations during tuberculosis treatment are not recommended. Chest x-rays performed during initial evaluation for tuberculosis, at 2-3 months after initiation of treatment for tuberculosis disease, and at completion of treatment, would be reasonable.

The Section of Epidemiology will provide reimbursement for chest x-rays only if authorization for payment has been **pre-approved** and if the patient does not have health insurance coverage which would pay for the x-ray. If pre-approved, total reimbursement for a chest x-ray and its interpretation will not exceed $40.00. The Section of Epidemiology will not pay for routine chest x-rays.

3. *Preventive Therapy*

Certain groups of people are known to be at high risk of developing tuberculosis. The benefits of preventive therapy with isoniazid in these individuals far outweigh the risks. However, isoniazid preventive therapy among these groups is **underused** in Alaska. These groups are as follows:

a. Household members and other close associates of potentially infectious tuberculosis cases.
b. Newly infected persons (those with tuberculin skin test conversion within the past two years, because the risk of developing disease is greatest during the 1-2 years immediately following infection).
c. Persons with significant reactions to tuberculin skin test and abnormal chest x-ray. Preventive therapy should be given to tuberculin skin test reactors with radiographic findings consistent with nonprogressive (previous) tuberculosis disease (negative bacteriology and stable parenchymal lesions).
d. Persons with significant reactions to a tuberculin skin test and who have any of the following clinical conditions which are associated with an increased risk of developing tuberculosis:
   - Silicosis.
   - Diabetes mellitus.
   - Prolonged treatment with adrenocorticosteroids.
   - Immunosuppressive therapy.
   - Some hematologic and reticuloendothelial dis-

eases, such as leukemia and Hodgkin's disease.

- Acquired immunodeficiency syndrome and persons with antibodies to the AIDS virus.
- End-stage renal disease.
- Clinical situations associated with substantial rapid weight loss or chronic undernutrition (e.g., intestinal bypass surgery, postgastrectomy state, chronic ulcer disease, malabsorption syndromes, etc.).

e. Tuberculin skin test reactors under 35 years of age with none of the above risk factors.

4. ***More Intensive Follow-up for Compliance with Treatment***

A major objective of the program is to insure that all patients complete recommended treatment for tuberculosis disease or infection. With the assistance of other public health personnel, the program will monitor and encourage patients' compliance with medical treatment (both preventive and therapeutic) and with clinical follow-up. Directly observed therapy will be employed, whenever possible, to assure patient compliance. Incentives (for example, provision of room and board during therapy) may be needed and will be provided for patients who resist treatment or whose lifestyles make compliance with treatment regimens difficult.

5. ***Increased Epidemiologic Investigations***

The Tuberculosis Control Program will conduct and/or coordinate epidemiologic investigations surrounding **all** potentially infectious cases of pulmonary tuberculosis (in order to identify persons infected by these cases) and newly infected individuals (in order to identify the source of infection and/or other infected persons).

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History of Medicine in Alaska

JOSEPH M. RIBAR, M.D.

Joseph Ribar is the son of Slovaks. His father immigrated to the United States from Stropkov, Ostrava (Czechoslovakia) at the age of sixteen. He worked first in the coal mines of Pennsylvania. Later riding the rails to Anaconda, Montana, he toiled in the copper mines. Finally, he continued west, becoming a railroad worker in Tacoma, Washington.

Joe Ribar's mother, Vera (Veronica) Catherine Ehnat's parents were from the Slovak area of Austro-Hungary. His mother was probably part white Russian. Dr. Ribar's parents met in Tacoma when the Ehnats moved there from Michigan.

Joe had wonderful parents and a tremendous childhood. His brother was 14 years older; his sister 12 years older. The Ribars were a solid Catholic family. Mother and father spoke and read Slavonian which Joe could understand. However, his father, as with so many immigrants, became so staunchly American that he once told Joe's sister, Mary: "You know English. Speak it in my house."

Joe went to the public school for six years. Then he attended the seventh and eighth grades at a Catholic school. He won a scholarship to Bellarmine High School, a Jesuit school, in Tacoma. The school was ten miles across town, and he used his bike until he wrecked it. His school provided a classical education with four years of Latin and two of Greek. There were few electives. Of course, there was one hour of religion taught each day. He was to be graduated at sixteen, valedictorian of his class.

He first attended the college, now university, of Puget Sound, working part time for 25 cents an hour at J.C. Penneys. He went to college in the mornings and worked afternoons.

Then he transferred full time to the University of Washington where he continued to work at Penneys and did odd jobs such as windows, floors and hauling gravel. He worked in a sorority house getting room and board. His job was as "houseboy": peeling potatoes, washing dishes and serving the girls at lunch. The war had begun, but he was classified 2-A as a pre-med student.

He was involved in the early work in genetics, begun with studies of the Midocontria Drosophylla, commonly known as the fruit fly. He has often wondered if he would have been successful in this field. But, he does not regret not pursuing it because he has felt more productive relating to people than he would have as a researcher.

Although he was accepted at five medical schools, he elected to stay close to home and attend the University of Oregon Medical School. At that time, the University of Washington had a law school; Oregon a medical school. They interchanged students in these disciplines. After receiving an M.D. in June, 1949, he had an internship at St. Joseph's Hospital, Tacoma.

Joe Ribar married Dorothy J. Karpach on September 14, 1946. Her grandmother had come from the same area in Czechoslovakia as Joe's father. Dorothy and Joe are both pure Slovak. They even went to the same church. This strong background means much to the Ribars.

At the end of June, 1950 the United States was engaged in the Korean War. Joe was called up in the naval reserve and was to serve as ship's doctor on a transport which sailed in and out of San Francisco to Yokohama. While this was much like being on a ferry, he did see the invasion of Inchon on September 15, 1950.

In 1952 he began a residency in neurosurgery under the auspices of the newly founded University of Washington Medical School at the Veterans' Administration Hospital in Seattle. By this time, Dorothy and he had three children, and he was making $200 a month. He was looking for a place to practice and through the American Medical Association discovered that a doctor was wanted in Fairbanks. He asked his wife about this, and she replied: "If you want to go, I guess I'll go."

He flew to Fairbanks in July 1953. It took three weeks to find a house. Then he and his family drove up over the Alaska Highway in August. The Hart Highway was being built, and there was approximately 150 miles of terrible road.

He was to begin practice with Dr. Haggland, an orthopedist, and Dr. Schiable, a surgeon. Joe thought of himself as a family practioner but fell into delivering babies, as many as 325 a year. Because the first pediatrician did not come to Fairbanks until 1962, he also did pediatrics. However, in the course of his practice, he pinned hips and operated on stomachs and gall bladders.

The polio epidemic of 1954 in Fairbanks made an impact. It was necessary to import equipment from Seattle. With regret, he recalls the death of a brilliant young university professor. He recalls there being many deaths.

He has witnessed the strange phenomenon of a patient with a minor illness which seemed to improve when hospitalized who nonetheless kept insisting he was going to die and did. Since the autopsy showed no cause of death, Joe has assumed that he scared himself to death. Dr Ribar claims that this has happened to him five or six times in his 35 year career.

Joe considers his pleasure in medicine to be involvement with his patients. The important continuity which exists in this relationship gives him great satisfaction. He is sorry that the new technology seems to have eliminated some of this.

He feels that it is important to participate in the community. He became part of the Fairbanks Medical Society and the Alaska State Medical Association. He has served on the Fairbanks City Council, 1958–59, and he was Mayor of Fairbanks, 1960–62.

Dr. Ribar became very active in the AMA. When Dr. Milo Fritz was delegate, Joe served as alternate. In 1974, he ran for vice-president of the AMA.

Pleasurably, he recalls his campaign for office aided and abetted by the late Dr. J. Ray Langdon, Dr. and Mrs. Kinn, Dr. Paul Isaac, and the Alaska State Medical Association. He believes that a party with typical Alaskan food, reindeer, king crab, salmon, moose, beaver, bear and porcupine, contributed to his eventual success. He served in 1974–75.

He notes that the name Ribar means fisher. When his parents were given wedding silver by fellow laundry workers of his mother, it had been marked with an "F." Even now Slovonian speaking patients will call him "Dr. Fisher."

The Ribars had five children, four boys and a girl. His oldest son was named Joseph. But, then when the youngest son was born on March 19th, St. Joseph's day, they had to name him James Joseph. It is a sorrow to Joe and Dorothy that this son was to die of a brain tumor at the age of 21.

After his son's death, Joe took some months off. He thought he was going to retire. However, he found himself taking refresher courses at the University of Colorado Medical School.

Also, he played golf which he has always enjoyed. One day on the third tee, he found himself wondering what his patients were doing. By the eleventh hole, he was finished with retirement.

Joe speaks of his good wife of 42 years who has helped with emotional support and reared their children. He has enjoyed their children. He says that he "doesn't want to live my life over. It might not be as good."

Joe Ribar reflects that retirement is the time in life when one should do exactly what he likes. However, he finds himself practicing six hours a day because that is what he enjoys. This is balanced by golf and bridge. Simply, Joseph Ribar likes people and his commitment to them.

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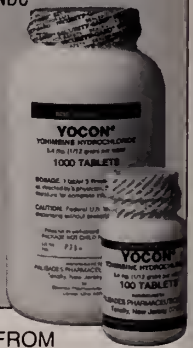

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News from the Alaska Public Health Association

President's Message

The New Year is bringing in a new look to the *ALPHA Newsletter*. At the November meeting, the Alaska Public Health Association Board voted to publish the *Newsletter* as a special section of *Alaska Medicine*, thus increasing the timeliness and frequency of your news from ALPHA. This change has two added benefits. It enhances our communication with the wider readership of *Alaska Medicine*, and it develops the function of *Alaska Medicine* as our official journal, which the membership reaffirmed at the April 1988, annual meeting. We hope you find this streamlining keeps you and our new readers better informed about vital issues affecting the public's health in Alaska.

Organizing to make a difference in the health of Alaskans was the focus of the ALPHA Leadership Retreat held at Alyeska Resort, September 9–11. Bill Dann, President of Professional Growth Systems, Inc., served as facilitator (and also became a new member). The dedicated and hardworking attendees devoted their weekend to setting ALPHA's course of action for the year.

This year's work plan is defined in the following four objectives and was endorsed by the Board.

- State Health Policy: to advocate for adoption of a State Health Policy by May 1, 1990.
- Injury Control: to secure additional funding in Alaska for injury control programs by October 1, 1989.
- Education: to increase yearly attendance at ALPHA educational sessions (excluding Congress) to 700 by June 1, 1989, and expand sites to seven (baseline 400).
- Membership: to increase individual membership to 225 and organizational membership to ten by May 1, 1989.

Here is a brief status report on these objectives:

• The development of a state health policy is being promoted through the Alaska Department of Health and Social Services. ALPHA surveyed selected people in the fields of injury control, mental health, and alcohol to develop a broad concept of how the policy might look. Sally Mead, Special Assistant to Myra Munson, Commissioner of Health and Social Services, has been charged with leading Division heads in a discussion on policy development. Tom Nighswander, ALPHA Health Issues Chairman, can be contacted for further information about this objective.

• The Injury Control issue is being spearheaded by Corlis Taylor of Bethel, newly appointed Board member, who is filling the vacancy created by Debra Plumb's resignation and departure from the state.

As the last newsletter articles pointed out, injuries are the leading cause of premature death of Alaskans, and one of the perceived needs is increased funding with which to deal with injuries. A first step is to establish a clearinghouse of injury control issues in the state. ALPHA is in a unique position to do this. A proposal was drafted and discussed on December 27 with Senator Stevens who is concerned about increasing efforts to reduce injuries. We will continue to develop this concept with his staff to obtain increased funding.

• Educational brown bag sessions held in Fairbanks, Anchorage, and Juneau have continued to be popular. Fairbanks has done a great job, thanks to Cheryl Kilgore's direction. Over 40 people attended the session on radon. It also received good press coverage.

We hope as many people as possible take advantage of these high quality offerings. They are excellent opportunities to learn about major public health issues in your community. (The Calendar of Events elsewhere in this issue lists them). If you would like to start one in your area for health workers and the public, contact Board member Cheryl Kilgore or Linda Vlastuin for details on how to do it.

• Finally, the membership goal will require the support of every existing member. We hope you have already renewed. I also hope you have asked your colleagues, students, and others interested in public health to join us. You don't need a degree in public health to be concerned about the health of Alaskans or to be a member of ALPHA. We hope to broaden our membership base this year and to become an even more effective organization. Have you considered an organizational membership in ALPHA for another group? We welcome this. Please contact Mark Johnson, Membership Chairman and our newest ALPHA Board member, for more information, recruitment suggestions, brochures, etc..

Other ideas were generated at the Fall Leadership Retreat. Your Board is striving for the most effective organization possible. Developing a more streamlined organization is one step in this process. The membership is being asked to vote on by-laws changes to make Board members chairpersons of the key standing committees. This will improve our communications and accountability. Active committees will continue to be our key to being an effective organization. All members are encouraged to be actively involved. Members can still serve as co-chairpersons with the new by-laws changes.

If passed, the by-laws revisions will also permit us to accept contributions (tax deductible) to further public health promotion in Alaska. Your vote is important.

I was saddened to receive Marty Dilly's resignation from the ALPHA Board. Marty has been a thoughtful and dedicated worker with ALPHA, and her contributions have been appreciated. However new career challenges are ahead for her as she leaves the public health field.

The Board held an all day meeting in Anchorage on December 3, monitoring work plans and progress on reaching our objectives. In addition, we responded to the Alaska Science and Technology Foundation's request for key public health issues requiring basic and applied research in Alaska. We identified application of injury control knowledge and evaluation of health promotion aimed at children as key issues.

Our next statewide gathering is just weeks away. The Health Congress Planning Committee under Susan Jones' leadership is developing an excellent program! We look forward to having you join us in Anchorage for "Reflections, Problems, and Promises: Planned Health through the Lifespan!"

Betsy Nobmann
President
Alaska Public Health Association

How to get what you want — Effective Lobbying

The following highlights were taken from the Anchorage ALPHA Brown Bag Lunch Presentation December 29, by Jana Varratti, Chair, Alaska Women's Lobby. For effective lobbying:

1. **Understand the process:**

   If you are new to the system, obtain a copy of "Legislative Process in Alaska" from the Legislative Affairs Office.

2. **Choose as your lobbyist or representative a person both knowledgeable and personable.**

   One or two representatives is usually better than a group, as communications have a tendency to become garbled with too many people involved.

3. **Do your homework.**

   Know both your subject and the people you need to address. Remember, legislative staff members are key players.

4. **Develop a plan of action before you begin.**

5. **Know both your support and your opposition.**

   It is often possible to co-opt your opposition by explaining their view before they can make their own pitch. Legislators will also appreciate your forthrightness.

6. **Be willing to do all the work.**

   Write as complete a draft of your proposed bill yourself as you can. It may also help to make yourself indispensable at a campaign time.

7. **Admit lack of financial backing if you have none.**

   Legislators are often impressed if you are spending your own money to make your point.

8. **Avoid gossip in Juneau. It can backfire.**

From the Membership Director,
"... Help boost membership by asking a friend to join ALPHA this year. An application is enclosed in the Newsletter."

Interim Health Care Commission Report

Executive Order No. 100, which Governor Steve Cowper signed on September 18, 1987, established the Governor's Interim Health Care Commission (HCC). The creation of the HCC reflects the concern of the Cowper administration that many Alaskans do not receive adequate health care and, that given the budgeting problems of the state, the current system may be difficult to maintain in the future. The Governor asked the HCC to recommend changes in laws, regulations and policies necessary to (1) reduce and/or contain the cost of health care in Alaska, and (2) increase the access to health care for Alaskans who lack the resources to obtain adequate health care.

Eleven members comprised the commission: Myra Munson, Commissioner of Health and Social Services; Senators Lloyd Jones and Willie Hensley; Representatives Johnny Ellis and Mark Boyer; and six public members appointed by the Governor (Sr. Barbara Haase, Sharon Jean, Mim Dixon, Thomas Senter, M.D., Judy Bush, Norma Lundy). Sharon Jean of Soldotna was selected as the chairperson.

Between November 1987 and July 1988, the Commission held eight meetings primarily to (1) identify causes for increasing health care costs, (2) review the role of state agencies and programs in providing essential health services, (3) analyze the delivery of services through state, federal, local and private health care programs, and (4) review the efforts of other states to manage costs while ensuring access to health care.

The Commission adopted 39 recommendations. Three pertain to providing adequate health care to children and pregnant women. The HCC found that Alaska has the highest post-neonatal death rate in the country and that, for some Alaskan women, prenatal care is unavailable. The Commission recommended (1) expanding Medicaid coverage to include more women and children, (2) developing a more extensive case management system for pregnant women (particularly those with high risk pregnancies), and (3) endorsing the Governor's Interim Commission on Children and Youth recommendations regarding comprehensive health screening, school nursing and public health nursing, adolescent survey, and fetal alcohol syndrome.

A second set of recommendations focuses on Alaskans' access to health care. The Commission found that an estimated 40,000 Alaskans are without health insurance. To provide health care for these individuals the Commission recommended (1) expansion of the General Relief Medical program, (2) the adoption of the Medicaid unemployed parent option so that two parent families may receive Medicaid coverage, (3) increased community efforts to provide health care to the indigent, and (4) the formation of a task force to develop health insurance plans for uninsured Alaskans.

A third set of recommendations supports efforts to develop, within Alaska, a continuum of long term care services which will help individuals avoid institutionalization. Specifically, the Commission recommended that the state develop a home and community based care system and a system of case management for individuals in need of care for the long term. Additional recommendations call for the development of affordable congregate housing for older Alaskans, as well as residential care options for individuals suffering from dementia.

The Commission also proposed changes in the ways in which the state purchases health care for its clients. Proposals include making the Medicaid Rate Commission advisory to the Department with regard to Medicaid reimbursement rates, applying a moratorium to the granting of certificates of need, and retaining pharmaceutical services as a Medicaid option. The Commission also suggested changes to payment policies of the Veterans Administration and the Indian Health Service so that services to beneficiaries of those agencies are not reduced. Finally, the Commission recommended increases in alcohol and tobacco taxes as a way to pay for expanded health care.

The last set of Commission recommendations is based on the belief that good health planning is critical to the state's efforts to manage health care costs. Good health planning examines financing, access, and quality and provides an understanding of the mix of the three. The Commission recommended that the state adopt a statement of health policy and make a commitment of sufficient resources to maintain a state health planning effort.

Jay Livey

Jay Livey is a Special Assistant to the Commissioner of Health and Social Services and served as a staffperson to the Governor's Interim Health Care Commission.

Opinion

National Health Service: The Solution to Chaos in Health Care

By Lawrence D. Weiss, PhD, MS
Assistant Professor, UAA Department of Sociology

There is an extraordinary trend in the progress of modern health care today in America. It is going backwards! Not too many years ago, the United States had one of the lowest infant mortality rates in the world. Today, the United States has a higher rate and is ranked twentieth among the world's nations. Hong Kong and Spain, for example, have lower infant mortality rates than the United States.

Health care costs are spiraling upwards out of control; yet there are now forty million Americans with no health insurance. Giant for-profit corporations are buying up hospitals across the United States at an alarming rate; yet they charge higher prices than non-profits, see fewer indigents, and dump about three million patients a year back onto the public or private non-profit sectors. The recent report by the Institute of Medicine, "The Future of Public Health," indicates that public health in America is in a state of complete chaos.

National or state health insurance plans have frequently been recommended as the answer to solve the nation's acute and worsening health care crisis. However, with these strategies huge profits would accrue to the health insurance companies, and the health care crisis would not be addressed. Health care costs would escalate even more rapidly. The public health crisis would in no way be addressed. Preventive health care strategies would continue to be ignored. Occupational and environmental health problems would proliferate unabated. Finally, the critical issues of malpractice and maldistribution of health care facilities would not be solved. Expanding health insurance is not the answer.

The answer is neither mystical nor unattainable. Implementation of Congressman Ron Dellums' (D-CA) bill, "The Health Service Act," would solve many of the most serious problems in American health care today. The Health Service Act would provide, without charge, comprehensive preventive and acute care to all persons in the United States. The National Health Service would be run on a democratic basis by elected boards of users and providers, beginning with each facility and extending all the way up to the level of national policy. All health care providers would be employees of the government. Health care would be rationally planned on a national basis, and financed by the hundreds of billions of dollars currently spent each year on health insurance and health care.

There would be no patient dumping, no problems of accessibility for the low income and uninsured, and no wildly escalating costs. Health care facilities and providers would be more rationally distributed. America's infant mortality rate would drop precipitously.

Internationally, National Health Service systems are quite common. In the United States there are a number of large organizations that already support a National Health Service, including the American Public Health Association representing tens of thousands of health workers. Would there be a difficult struggle? Yes, absolutely. Like the struggles for the eight hour workday, woman suffrage, and civil rights, the struggle for a National Health Service would be difficult, but successful.

Note: The American Public Health Association has officially supported the concept of a National Health Service since 1976.

The opinions expressed in this column are those of the author and do not necessarily reflect those of ALPHA. To submit an article to this column, send two double-spaced, typed pages to Toni Niesen, 17741 Mountainside Village Dr., Anchorage, Alaska 99516, or call 343-4773.

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Hypotension. Excessive hypotension is rare in uncomplicated hypertensive patients treated with VASOTEC alone. Heart failure patients given VASOTEC commonly have some reduction in blood pressure, especially with the first dose, but discontinuation of therapy for continuing symptomatic hypotension usually is not necessary when dosing instructions are followed, caution should be observed when initiating therapy. (See DOSAGE AND ADMINISTRATION.) Patients at risk for excessive hypotension, sometimes associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death, include those with the following conditions or characteristics: heart failure, hyponatremia, high-dose diuretic therapy, recent intensive diuresis or increase in diuretic dose, renal dialysis, or severe volume and/or salt depletion of any etiology It may be advisable to eliminate the diuretic (except in heart failure patients), reduce the diuretic dose, or increase salt intake cautiously before initiating therapy with VASOTEC in patients at risk for excessive hypotension who are able to tolerate such adjustments. (See PRECAUTIONS, *Drug Interactions* and ADVERSE REACTIONS.) In patients at risk for excessive hypotension, therapy should be started under very close medical supervision and such patients should be followed closely for the first two weeks of treatment and whenever the dose of enalapril and/or diuretic is increased. Similar considerations may apply to patients with ischemic heart disease or cardiovascular disease in whom an excessive fall in blood pressure could result in a myocardial infarction or cerebrovascular accident

If excessive hypotension occurs, the patient should be placed in supine position and, if necessary, receive an intravenous infusion of normal saline. A transient hypotensive response is not a contraindication to further doses of VASOTEC, which usually can be given without difficulty once the blood pressure has stabilized If symptomatic hypotension develops, a dose reduction or discontinuation of VASOTEC or concomitant diuretic may be necessary.

Neutropenia/Agranulocytosis. Another ACE inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression, rarely in uncomplicated patients but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease. Available data from clinical trials of enalapril are insufficient to show that enalapril does not cause agranulocytosis at similar rates. Foreign marketing experience has revealed several cases of neutropenia or agranulocytosis in which a causal relationship to enalapril cannot be excluded Periodic monitoring of white blood cell counts in patients with collagen vascular disease and renal disease should be considered.

Precautions: *General: Impaired Renal Function:* As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including VASOTEC, may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine were observed in 20% of patients. These increases were almost always reversible upon discontinuation of enalapril and/or diuretic therapy In such patients, renal function should be monitored during the first few weeks of therapy.

Some patients with hypertension or heart failure with no apparent preexisting renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when VASOTEC has been given concomitantly with a diuretic. This is more likely to occur in patients with preexisting renal impairment. Dosage reduction and/or discontinuation of the diuretic and/or VASOTEC may be required.

Evaluation of patients with hypertension or heart failure should always include assessment of renal function. (See DOSAGE AND ADMINISTRATION.)

Hyperkalemia Elevated serum potassium (> 5.7 mEq/L) was observed in approximately 1% of hypertensive patients in clinical trials. In most cases these were isolated values which resolved despite continued therapy. Hyperkalemia was a cause of discontinuation of therapy in 0.28% of hypertensive patients In clinical trials in heart failure, hyperkalemia was observed in 3 8% of patients, but was not a cause for discontinuation

Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with VASOTEC (See *Drug Interactions.*)

Surgery/Anesthesia: In patients undergoing major surgery or during anesthesia with agents that produce hypotension, enalapril may block angiotensin II formation secondary to compensatory renin release If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion.

Information for Patients:

Angioedema Angioedema, including laryngeal edema, may occur especially following the first dose of enalapril. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to take no more drug until they have consulted with the prescribing physician.

Hypotension. Patients should be cautioned to report lightheadedness especially during the first few days of therapy If actual syncope occurs, the patients should be told to discontinue the drug until they have consulted with the prescribing physician.

All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduction in fluid volume. Other causes of volume depletion such as vomiting or diarrhea may also lead to a fall in blood pressure, patients should be advised to consult with the physician.

Hyperkalemia Patients should be told not to use salt substitutes containing potassium without consulting their physician

Neutropenia Patients should be told to report promptly any indication of infection (e g , sore throat, fever) which may be a sign of neutropenia

NOTE As with many other drugs, certain advice to patients being treated with enalapril is warranted This information is intended to aid in the safe and effective use of this medication It is not a disclosure of all possible adverse or intended effects

Drug Interactions

Hypotension: Patients on Diuretic Therapy Patients on diuretics and especially those in whom diuretic therapy was recently instituted may occasionally experience an excessive reduction of blood pressure after initiation of therapy with enalapril. The possibility of hypotensive effects with enalapril can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with enalapril. If it is necessary to continue the diuretic, provide close medical supervision after the initial dose for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and DOSAGE AND ADMINISTRATION.)

Agents Causing Renin Release. The antihypertensive effect of VASOTEC is augmented by antihypertensive agents that cause renin release (e.g., diuretics).

Other Cardiovascular Agents: VASOTEC has been used concomitantly with beta-adrenergic-blocking agents, methyldopa, nitrates, calcium-blocking agents, hydralazine, prazosin, and digoxin without evidence of clinically significant adverse interactions

Agents Increasing Serum Potassium: VASOTEC attenuates potassium loss caused by thiazide-type diuretics Potassium-sparing diuretics (e g , spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium. Therefore, if concomitant use of these agents is indicated because of demonstrated hypokalemia, they should be used with caution and with frequent monitoring of serum potassium. Potassium-sparing agents should generally not be used in patients with heart failure receiving VASOTEC

Lithium: A few cases of lithium toxicity have been reported in patients receiving concomitant VASOTEC and lithium and were reversible upon discontinuation of both drugs. Although a causal relationship has not been established, it is recommended that caution be exercised when lithium is used concomitantly with VASOTEC and serum lithium levels should be monitored frequently

Pregnancy—Category C: There was no fetotoxicity or teratogenicity in rats treated with up to 200 mg/kg/day of enalapril (333 times the maximum human dose) Fetotoxicity, expressed as a decrease in average fetal weight, occurred in rats given 1200 mg/kg/day of enalapril but did not occur when these animals were supplemented with saline Enalapril was not teratogenic in rabbits However, maternal and fetal toxicity occurred in some rabbits at doses of 1 mg/kg/day or more. Saline supplementation prevented the maternal and fetal toxicity seen at doses of 3 and 10 mg/kg/day, but not at 30 mg/kg/day (50 times the maximum human dose).

Radioactivity was found to cross the placenta following administration of labeled enalapril to pregnant hamsters

There are no adequate and well-controlled studies in pregnant women VASOTEC® (Enalapril Maleate, MSD) should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers. Milk in lactating rats contains radioactivity following administration of ^{14}C enalapril maleate It is not known whether this drug is secreted in human milk Because many drugs are secreted in human milk, caution should be exercised when VASOTEC is given to a nursing mother.

Pediatric Use: Safety and effectiveness in children have not been established

Adverse Reactions: VASOTEC has been evaluated for safety in more than 10,000 patients, including over 1000 patients treated for one year or more. VASOTEC has been found to be generally well tolerated in controlled clinical trials involving 2987 patients.

Hypertension: The most frequent clinical adverse experiences in controlled trials were: headache (5.2%), dizziness (4 3%), and fatigue (3%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in controlled clinical trials were: diarrhea (1.4%), nausea (1 4%), rash (1.4%), cough (1.3%), orthostatic effects (1.2%), and asthenia (1.1%).

Heart Failure: The most frequent clinical adverse experiences in both controlled and uncontrolled trials were dizziness (7.9%), hypotension (6 7%), orthostatic effects (2 2%), syncope (2 2%), cough (2.2%), chest pain (2.1%), and diarrhea (2.1%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in both controlled and uncontrolled clinical trials were: fatigue (1.8%), headache (1.8%), abdominal pain (1 6%), asthenia (1.6%), orthostatic hypotension (1.6%), vertigo (1.6%), angina pectoris (1.5%), nausea (1.3%), vomiting (1.3%), bronchitis (1.3%), dyspnea (1 3%), urinary tract infection (1.3%), rash (1.3%), and myocardial infarction (1.2%).

Other serious clinical adverse experiences occurring since the drug was marketed or adverse experiences occurring in 0.5% to 1% of patients with hypertension or heart failure in clinical trials in order of decreasing severity within each category:

Cardiovascular: Myocardial infarction or cerebrovascular accident, possibly secondary to excessive hypotension in high-risk patients (see WARNINGS, *Hypotension*), cardiac arrest; pulmonary embolism and infarction; rhythm disturbances, atrial fibrillation; palpitation.

Digestive Ileus, pancreatitis, hepatitis or cholestatic jaundice, melena, anorexia, dyspepsia, constipation, glossitis

Nervous/Psychiatric. Depression, confusion, ataxia, somnolence, insomnia, nervousness, paresthesia

Urogenital: Renal failure, oliguria, renal dysfunction (see PRECAUTIONS and DOSAGE AND ADMINISTRATION), prostate hypertrophy

Respiratory: Bronchospasm, rhinorrhea, asthma, upper respiratory infection.

Skin: Herpes zoster, pruritus, alopecia, flushing, photosensitivity

Other: Muscle cramps, hyperhidrosis, impotence, blurred vision, taste alteration, tinnitus.

A symptom complex has been reported which may include fever, myalgia, and arthralgia, an elevated erythrocyte sedimentation rate may be present. Rash or other dermatologic manifestations may occur These symptoms have disappeared after discontinuation of therapy.

Angioedema Angioedema has been reported in patients receiving VASOTEC (0 2%). Angioedema associated with laryngeal edema may be fatal If angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with VASOTEC should be discontinued and appropriate therapy instituted immediately. (See WARNINGS.)

Hypotension: In the hypertensive patients, hypotension occurred in 0.9% and syncope occurred in 0 5% of patients following the initial dose or during extended therapy Hypotension or syncope was a cause for discontinuation of therapy in 0.1% of hypertensive patients. In heart failure patients, hypotension occurred in 6 7% and syncope occurred in 2.2% of patients Hypotension or syncope was a cause for discontinuation of therapy in 1.9% of patients with heart failure. (See WARNINGS.)

Clinical Laboratory Test Findings:

Serum Electrolytes: Hyperkalemia (see PRECAUTIONS), hyponatremia

Creatinine, Blood Urea Nitrogen: In controlled clinical trials, minor increases in blood urea nitrogen and serum creatinine, reversible upon discontinuation of therapy, were observed in about 0.2% of patients with essential hypertension treated with VASOTEC alone Increases are more likely to occur in patients receiving concomitant diuretics or in patients with renal artery stenosis. (See PRECAUTIONS.) In patients with heart failure who were also receiving diuretics with or without digitalis, increases in blood urea nitrogen or serum creatinine, usually reversible upon discontinuation of VASOTEC and/or other concomitant diuretic therapy, were observed in about 11% of patients. Increases in blood urea nitrogen or creatinine were a cause for discontinuation in 1 2% of patients.

Hemoglobin and Hematocrit: Small decreases in hemoglobin and hematocrit (mean decreases of approximately 0.3 g % and 1 0 vol %, respectively) occur frequently in either hypertension or heart failure patients treated with VASOTEC but are rarely of clinical importance unless another cause of anemia coexists. In clinical trials, less than 0.1% of patients discontinued therapy due to anemia.

Other (Causal Relationship Unknown): In marketing experience, rare cases of neutropenia, thrombocytopenia, and bone marrow depression have been reported

Liver Function Tests: Elevations of liver enzymes and/or serum bilirubin have occurred

Dosage and Administration: *Hypertension:* In patients who are currently being treated with a diuretic, symptomatic hypotension occasionally may occur following the initial dose of VASOTEC. The diuretic should, if possible, be discontinued for two to three days before beginning therapy with VASOTEC to reduce the likelihood of hypotension (See WARNINGS.) If the patient's blood pressure is not controlled with VASOTEC alone, diuretic therapy may be resumed.

If the diuretic cannot be discontinued, an initial dose of 2.5 mg should be used under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions.*)

The recommended initial dose in patients not on diuretics is 5 mg once a day Dosage should be adjusted according to blood pressure response The usual dosage range is 10 to 40 mg per day administered in a single dose or in two divided doses. In some patients treated once daily, the antihypertensive effect may diminish toward the end of the dosing interval In such patients, an increase in dosage or twice-daily administration should be considered. If blood pressure is not controlled with VASOTEC alone, a diuretic may be added

Concomitant administration of VASOTEC with potassium supplements, potassium salt substitutes, or potassium-sparing diuretics may lead to increases of serum potassium (see PRECAUTIONS)

Dosage Adjustment in Hypertensive Patients with Renal Impairment: The usual dose of enalapril is recommended for patients with a creatinine clearance >30 mL/min (serum creatinine of up to approximately 3 mg/dL). For patients with creatinine clearance ≤30 mL/min (serum creatinine ≥3 mg/dL), the first dose is 2.5 mg once daily The dosage may be titrated upward until blood pressure is controlled or to a maximum of 40 mg daily

Heart Failure: VASOTEC is indicated as adjunctive therapy with diuretics and digitalis. The recommended starting dose is 2.5 mg once or twice daily. After the initial dose of VASOTEC, the patient should be observed under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions.*) If possible, the dose of the diuretic should be reduced, which may diminish the likelihood of hypotension. The appearance of hypotension after the initial dose of VASOTEC does not preclude subsequent careful dose titration with the drug, following effective management of the hypotension. The usual therapeutic dosing range for the treatment of heart failure is 5 to 20 mg daily given in two divided doses. The maximum daily dose is 40 mg Once-daily dosing has been effective in a controlled study, but nearly all patients in this study were given 40 mg, the maximum recommended daily dose, and there has been much more experience with twice-daily dosing In addition, in a placebo-controlled study which demonstrated reduced mortality in patients with severe heart failure (NYHA Class IV), patients were treated with 2.5 to 40 mg per day of VASOTEC, almost always administered in two divided doses. (See CLINICAL PHARMACOLOGY, *Pharmacodynamics* and *Clinical Effects.*) Dosage may be adjusted depending upon clinical or hemodynamic response (See WARNINGS.)

Dosage Adjustment in Heart Failure Patients with Renal Impairment or Hyponatremia: In heart failure patients with hyponatremia (serum sodium <130 mEq/L) or with serum creatinine >1.6 mg/dL, therapy should be initiated at 2.5 mg daily under close medical supervision. (See DOSAGE AND ADMINISTRATION, *Heart Failure*, WARNINGS, and PRECAUTIONS, *Drug Interactions.*) The dose may be increased to 2.5 mg b.i.d., then 5 mg b.i.d. and higher as needed, usually at intervals of four days or more, if at the time of dosage adjustment there is not excessive hypotension or significant deterioration of renal function The maximum daily dose is 40 mg

For more detailed information, consult your MSD representative or see Prescribing Information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, PA 19486

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News in Brief

Reducing injuries in the home to children under four years of age will be the purpose of an eighteen month state grant awarded to the Health Education Section of the Municipality of Anchorage, Department of Health and Human Services. Under terms of the grant, 62 residences in a public housing project (Loussac Manor in Anchorage) will be fitted with home safety devices. Injury prevention, training and education will also be provided. Upon completion of the project, injury statistics will be compared with those prior to initiation of the safety program.

★ ★ ★

Sweeping changes to the Indian Self Determination Act (PL 93-638) will occur as a result of amendments set forth in PL 100-472. This law requires that new regulations reflecting these amendments be written within ten months. The most significant feature of the amendment will be removing ISDA contracts from the control of federal acquisition regulations and requiring instead that 638 contractors be managed in a manner similar to states. The new regulations will be published in the Federal Register for comment in May 1989.

★ ★ ★

Merger: Anchorage Center for Families (ACF) is the title for the newly consolidated Center for Children and Parents, Intermission, and Family Connection.

★ ★ ★

Maniilaq has recently assumed management of the Kotzebue service unit of Indian Health Service. This will include Kotzebue hospital.

★ ★ ★

ALPHA membership voted in favor of changing the bylaws. The new bylaws are effective February 3, 1989.

DEC will be subcontracting audits for small businesses regarding hazardous materials used and generated with emphasis on waste reduction. Representative Kay Brown plans to introduce legislation to make hazardous waste reduction the primary management tool.

★ ★ ★

The Aleutian East Borough, formed one year ago, is currently reviewing health needs in the communities of Akutan, Sand Point, King Cove, Cold Bay, False Pass, and Nelson Lagoon. The Borough does not possess health powers but recognizes that the population of approximately 2500 experiences difficulties obtaining health care. The major challenges identified in a recent study by Bill Dann of Professional Growth Systems are as follows:

1. A regional hub for transportation or services is lacking. Residents experience logistic difficulties, including having to fly to Anchorage as an intermediate stop in order to travel from one Aleutian community to another.

2. Responsibility for health care is dispersed. Indian Health Services and the Aleutian Pribilof Island Association serve natives only and exclude mid-level health services. The state historically has supported clinics in Sand Point, King Cove and Cold Bay only.

3. Population varies with season. During fishing season, the population of some communities doubles, and trauma rates increase.

The focus of the study was to determine what can be done to provide health services to the entire population and to seek community input and support. The Borough Assembly will review the entire report February 9, and will decide what action should be taken. If assuming health powers is a necessary part of the plan, an election will be required for authorization.

WANTED

ALPHA is in need of a volunteer to act as Archivist. We have accumulated records over the years which need to be condensed and streamlined. The Board has authorized two file cabinets to be housed at the Alaska Health Project, 411 W 7th St., Anchorage for our official records. If you have the time and like things in order, here is your opportunity to be of help.

Please contact Betsy Nobmann 257-1316.

CALENDAR OF EVENTS

ISSUES AND STRATEGIES IN THE MANAGEMENT AND CONTROL OF TUBERCULOSIS TODAY

March 15, 1989
5 p.m.-9:15 p.m.
Dimond High School Theater,
2909 88th Ave. Anchorage

Speaker: Michael D. Iseman, MD
Director, Microbacteriology
National Jewish Center for Immunology
and Respiratory Medicine,
Denver, Colorado.

For information call:
Vicky Webb — MOA TB Clinic 343-4601
Deborah Williams — Lung Association
276-5864

ALPHA BROWN BAG LUNCHES

1231 Gambell Street
Anchorage, Third Floor
Conference Room, 1200-1:00 p.m.

March 30, 1989
Nutrition in Rural Alaska; Nutritional Changes in the Native Diet-Implications for Health Professionals — Betsy Nobmann, Chief, Nutrition Section, Alaska Native Health Service

April 27, 1989
SIDS Update/Current Research, New Challenges to an Old Problem — Cindy Schraer, MD, Alaska Native Health Service

May 25, 1989
Teenage Runaways in Anchorage-Alaska Takes a Positive Step Forward — Jane Barnes, Program Coordinator, Covenant House Project, Anchorage

Barbara Berger Award for Excellence in Health Education

The Alaska Health Education Consortium (AHEC) is seeking nominations for "The Barbara Berger Award for Excellence in Health Education."

This award is to recognize outstanding dedication and vision in health education and promotion in the State of Alaska.

Criteria for Nomination:

1) Open to individuals, agencies, and programs in Alaska.
2) Contributes to the advancement of health education and promotion in Alaska.
3) Recognized by their community for their dedication in promoting health and wellness.
4) Demonstrates a personal commitment to the advancement to individual health, wellness and self-responsibility through creativity, imagination and a sense of humor in working with people.
5) Through example, serves as an inspiration others in pursuit of excellence.

ALPHA Executive Board & Committees 1988–89

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Frank Pauls, Board/By–Laws Committee Chairperson	3431 Cottonwood, Anchorage, AK 99508	H – 274-9930	W – 274-9330
Carl Hild, Board/Treasurer	705B West 13th Avenue, Anchorage, AK 99501	H – 274-4453	W – 276-2864
Elaine McKenzie, Board/Awards and Nominations Committee Chairperson	800 Airport Road, Fairbanks, AK 99701	H – 479-6151	W – 452-1776
Mark Johnson, Board/Membership Chairperson	P.O. Box H–06C, Juneau, AK 99811	H – 789-2238	W – 465-3027
Bert Hall, Affiliate Representative, APHA	Box 378, Anchorage, AK 99510	H – 243-1778	W – 343-6718
Tom Kovalesky, Representative, CPHA	ANMC, 255 Gambell, Anchorage, AK 99501	H –	W – 257-1317
Jan Wills, Continuing Education Committee Chairperson	15401 Wills Way, Anchorage, AK 99516	H –	W – 343-4800
Duff Pfanner, Finance Committee Chairperson	1822 Buccaneer Place, Anchorage, AK 99501	H – 278-1057	W – 257-1145
Patty Owen, Juneau Local Representative	3245 Hospital Drive, Juneau, AK 99801	H – 586-2482	W – 463-4000
Toni Niesen, Publicity Chairperson/Newsletter Editor	AMHD, P.O. Box 196650, Anchorage, AK 99519	H – 345-6722	W – 343-4773
Mary Ann Field, Assistant Newsletter Editor	200 West 34th, #120, Anchorage, AK 99503	H – 345-2695	W – 258-4545
Jeanne Pontti, Board	Ketchikan Health Center, Ketchikan, AK 99901	H –	W – 225-4350

If you haven't had a mammogram, you need more than your breasts examined.

A mammogram is a safe, low-dose X-ray that can detect breast cancer before there's a lump. In other words, it could save your life and your breast.

If you're a woman over 35, be sure to schedule a mammogram. Unless you're still not convinced of its importance.

In which case, you may need more than your breasts examined.

Find the time.
Have a mammogram.

AMERICAN CANCER SOCIETY®

Give yourself the chance of a lifetime.

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Job Title ________ Employed by ________

_____ $50 annual dues – individual

_____ $1000 lifetime – individual

_____ $150 annual – organization

_____ $15 annual – student, retired and unemployed

Please list your area(s) of interest: Public health issues; Budget & finance; Continuing education; Special programs; Newsletter & publicity; Annual awards; Membership; Nominating; Constitution; By–laws & History . . .

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Make checks payable & mail to:

Alaska Public Health Association
PO Box 9–1825
Anchorage, Alaska 99509

Glimpses of Alaskan Medical History

Edited by Robert Fortuine, M.D.

Surgical Techniques of the Koniag (1802–1807)

One of the most useful early ethnographies of the Koniag, or Natives of Kodiak Island, was prepared by a young Russian naval officer named Gavriil Davydov, who entered the service of the Russian–American Company in 1802. He and a fellow officer named N. A. Khvostov travelled across Siberia and finally reached Kodiak late that summer. The two spent the winter at the Russian settlement, where Davydov spent much of his leisure time studying the manners and customs of the Koniag.

Returning to St. Petersburg the following summer, the two retraced their long journey back to Alaska. In the spring of 1806 they made a voyage with Count Nikolai Rezanov from Sitka to Spanish California to buy food for the fledgling colony at that time threatened with starvation. Later, after several unusual adventures in the Far East, Davydov and Khvostov made their way back to St. Petersburg. In 1808, Davydov drowned in the Neva River as he was returning home from a party with a couple of old friends from Alaska.

Davydov had already begun to write his memoirs at the time of his death, but had only finished the account of his first journey to Alaska and back. A number of ethnographic notes of great value, including those on diseases and healing, were collected from letters and published by a friend as Part II of his narrative.

"It appears that the most prevalent illness amongst the islanders is boils, which are dangerous everywhere that fish forms the main part of the diet together with fat and other raw foods. Sometimes the boils do not burst but spread and sometimes cover the whole of the back. In such cases the skin is lanced with a sharp shell and the pus is sucked out. But if the core is very deep then they take a hard, sharpened stone set in wood so that the point of the stone is sticking out; this they place on the core or center of the boil up to the very handle and they twist it about. The pain produced by this action can well be imagined. When the pus is reached in this manner it is sucked out and and the remainder comes out later of its own accord.

"If the back has been painful for a long while then the sufferer is made to sit on a table or some kind of plank and two such stones are attached to the base of his back, either side of the backbone. Then he is told to lie back and to remain lying down until the blood stops flowing.

"At the onset of any illness whatever the first precaution an islander takes is to eat as little as possible—even cases of venereal disease this is almost the only cure. . . .

"In the case of some illnesses the Koniagas let blood from various parts of the body; such as the arms, legs, forehead, etc. But their method of letting the blood is very effective. They cut the skin near the vein from which they wish to take the blood, then they place below the vein a needle or sharpened bone and let the blood with a sharp shell. The office of barber is for the most part occupied by the women. For shortness of breath the Koniagas let blood from below the chin.

"They perform some surgery. If they are wounded, for example, they will cut out the ball or arrow-head. They will also set broken arms, legs and so forth. I saw one islander, chopping wood, cut himself across the stomach; it was immediately sewn up and he quickly recovered.

"One islander had suffered for some time from stones but could find no one who would cut the stones out of him. In the end one old man offered to do it and although, after the operation was performed on him, the Koniaga lay for a week in such condition that he was barely distinguishable from a corpse, in the end he gradually began to get well and eventually he recovered completely.

"Many Russians have assured me that they have seen with their own eyes how the islanders remove cataracts from their eyes. But once this method has been described I leave everyone free to believe it or not, because as I have not been a witness to it I cannot confirm its veracity. The Koniagas are said to take a large louse, to which they attach a hair. This is then lowered into the person's eye and the hair is jerked so that the louse will grasp the cataract.

"Fever is completely unknown to the Konigas, and even the Russians very rarely go down with it.

"I have seen several savages suffering from sores, mostly on the legs and feet. One had had them for seven years; all of the right leg was covered in sores and in between them the flesh was raw. All those infected with this disease give off an unbearable and noxious odor."

REFERENCE

Davydov, GI. *Two Voyages to Russian America, 1802–1807.* Trans. by Colin Bearne. Ed. by Richard A. Pierce. Kingston, Ontario: Limestone Press, 1977. Pp. 177–179.

ALASKA STATE MEDICAL ASSOCIATION
and
FEDERAL AVIATION ADMINISTRATION
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44th ANNUAL CONVENTION
JUNE 6 - 11, 1989

A Whole Physician in a Healing Universe

Registration/Information

Alaska State Medical Association
Jeanette McCart, Executive Assistant
2401 E. 42nd Avenue, Suite 104
Anchorage, Alaska 99508
Phone (907) 562-2662

ASMA/FAA cosponsored meeting.

Registration Fee: $250.00 for Non-member physicians. This fee entitles physicians to attend all CME sessions. No fee is required for ASMA members or FAA registrants.

Please register prior to April 15, 1989.

Location & Date of Meeting

Sheldon Jackson College
Sitka, Alaska
June 6-11, 1989

Continuing Medical Education

Over 25 hours of scientific medical education will be provided by nationally recognized physicians.

Accommodations

Over 300 rooms have been reserved at special rates in Sitka, all within walking distance to Sheldon Jackson College and the Centennial Building.

Activities

Excitement will be high in Sitka! Attendees can enjoy the Sitka Music Festival, New Archangel Dancers, relax while cruising to an island for a salmon/halibut bake, tour historic Russian America, meander through Totem National Park, and for the adventureous, run for fun with Alaska's fastest (and not so fast). The grand finale to five fun-packed days will be the President's Dinner Ball.

Family Fun

A full slate of spouse and children's activities are being planned by the ASMA Auxiliary. Babysitting will be available.

Transportation

Alaska Airlines provides daily jet service from Seattle, Anchorage, and other Alaskan cities. Phone 800-426-0333 for reservations. For local info, check phone book.

Ferry service is available from Seattle and S.E. Alaska. Phone (800) 642-0066. Shuttle service and rental cars are available in Sitka.

Deposit/Cancellation

Non-member physicians must send $50 preregistration fee by April 15. Full refunds will be made upon request prior to May 1, 1989. No refund thereafter.

Schedule of Events

Tuesday - June 6, 1989

8:30-4:00 p.m. - AME Workshop
1:30 p.m.- ASMA Council Meeting
3:00-5:00 p.m.- House of Delegates
5:00 p.m.- Dinner

Wednesday - June 7, 1989

8:00-9:00 a.m. - Breakfast
Keynote speaker
9:00-12:00 noon-Scientific Session
1:30-5:00 p.m.-Scientific Session
5:15-6:15 p.m.-Exhibitors' Reception
6:30 p.m.-Russian Dinner
w/Entertainment
Survival Training throughout Day

Thursday - June 8, 1989

8:00-12:00 noon-Scientific Session
12:15-1:15 p.m.-Lunch with Speaker
1:30-5:00 p.m.-Scientific Session
5:00 p.m.-Dinner on your own
7:00 p.m.- Sitka Music Festival
(tentative private performance)
Survival Training Throughout Day

Friday - June 9, 1989

8:00-11:45 a.m.- Scientific Session
12:00 noon - Lunch w/ Speaker
1:30- 4:45 p.m.- Scientific Session
5:15 p.m.-Cruise to Salmon/Halibut
Bake on Island

Saturday - June 10, 1989

6:30 a.m.- AAFP Fun Run
8:00 a.m -Breakfast - Charles Chappell
Associate Director for
Science, NASA
8:50-9:45 a.m - Scientific Session
10:15-11:45a.m.-Panel Discussion
12:00 noon - Lunch w/ Speaker
1:30-5:00 p.m. - House of Delegates
6:30 p.m. - President's Banquet&Ball
Keynote Speaker -
C. Everett Koop (tentative)
Entertainment & Awards

Sunday - June 11, 1989

9:00 a.m.- Council Meeting

REGISTRATION FORM

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President's Page

"Spare No Expense, Doc. When it comes to my health, money is no object!"

When a doctor hears that from a patient he can be pretty certain of two things—he is not going to be paid, and if there is a bad outcome he very likely will be sued. Fortunately we do not hear that very often from individual patients. But, there are distressing signs that the general public is taking an attitude which closely corresponds to this position.

There seems to be a growing feeling in many people that every person in this country should be offered the most expensive and most advanced treatment available for whatever the problem, even if there is little hope for prolonging length of life or increasing the ability to enjoy it. If treatment is available and the patient wants it, he should have it. Furthermore, the direct cost of the treatment to the patient should not cause him any great financial distress. The cost should be born by his insurance carrier or by a program piad for by the government–city, state or federal.

If this sounds like an overblown statement, consider the public condemnation of the State of Oregon's decision last July to pay for no further heart, liver or bone marrow transplants from Medicaid funds and instead to divert this money to prenatal care. Of course, prior to the Oregon decision, everyone remembers the national media attention and the outpouring of money, including a check from President Reagan, which went to the child who needed a liver transplant and could not afford to pay the hospital bill. Incidentally, the ocurt recently ruled that the mother was allowed to keep the Mercedes that she bought with the donated money after her child's death. No, it is not an exaggeration to say that a lot of people expect everyone should have whatever treatment he wants regardless of cost.

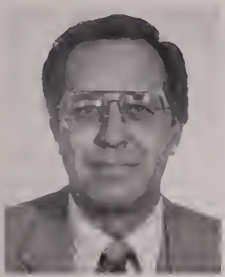
If this is the public expectation, what is the reality?

If we look at countries similar to the United States (England, Sweden, Canada) which provide health care at little or no direct cost to the individual, it is apparent that one way these countries keep the expense down is that they provide less care. Patients do not necessarily get what they, or their doctors, think would be ideal. They get what the system can afford. In the United States, for example, 20 percent of babies are born by Caesarian seciton compared to less than 10 percent in England. In the United States 61 per every 100,000 persons have coronary artery by–pass surgery compared to 6 per 100,000 persons in England. Without debating whether this is good or bad, this is the way it is. Would our patients be willing to have their choices of treatment limited by fiscal as well as medical considerations? The reality is that if the federal government, followed closely by large corporations and their insurers, proceeds in the direction it is currently taking, choices of treatment increasingly will be limited by financial considerations.

We, the physicians, may not be able to bring about a change in this direction, but we have an obligation to educate our patients to what lies ahead. We need to let our patients know that less expensive medicine is "less" medicine, not the same medicine for less money. We need to make certain that our patients realize that just cutting back on what doctors and hospitals charge will not make it possible to provide the newest and the most expensive treatment to everyone. People will have to accept that the most expensive, state–of–the–art, treatment is simply not always affordable. This will not be easy for many Americans to accept. Those who support government subsidised medical care would like us to believe that our country is wealthy enough so that there should be no financial restrictions on medical care. We are in the process of rapidly finding out that this is not true.

Irvin A. Rothrock, M.D.
President
Alaska State Medical Association

Executive Privilege



Recently, a letter was mailed to each physician in Alaska announcing and explaining the creation of the Health Care Coalition of Alaska (HCC). Although this group has been meeting monthly since April of 1987, the associations and societies represented have recently approved the bylaws and operating procedures that now formalize this Coalition.

Members of the Coalition are Tom Kelly, past-president of the Alaska Public Health Association, Harlan Knudson, executive director of the Health Association of Alaska, Martha Dearborn, executive director of the Alaska Dental Society, Kay Lahdenpera, past-president of the Alaska Nurses Association, Jim Tanner, past-president of the Alaska Academy of Physicians Assistants, and Bill Davnie, past-president of the Alaska Pharmaceutical Association.

The seed for the Coalition was planted as a result of dialogue within the provider community about the developing problem of health care access and affordability for our citizens. The first meeting in April of 1987 addressed our concern for the medically indigent and the need for a health policy for the State of Alaska. During those 1987 meetings we attempted to create the forum to exchange our views on current issues and reach a consensus on a statement of purpose.

As the stewards of health care, it was important that our statement of purpose reflect not only our homogeneity, but our desire to be patient advocates. As a result, our Statement of Purpose reads, "The Health Care Coalition of Alaska is committed to the betterment of the publics health by developing, monitoring, and distributing information; by advocating for health-related rights, responsibilities, and issues; and by representing the professions as a whole in an ethical and responsible manner which would bring honor to the health care profession. This is to be done in a manner that protects the autonomy of the member organizations, improves the practice of health providers, and builds the public trust and confidence in the competence and reliability of the individual profession. The coalition seeks to enhance the activities of member organizations through cooperative and unified activities."

In early 1988, we addressed issues that dealt with the VA Hospital, Interim Commission on Health Care, tort, vaccine indemnification, state and federal health legislation, AIDS, professional impairment, PADS, and co-sponsored a pain symposium.

In addition, a personal relationship was being developed with our congressional delegation. During the recent session of congress, the delegation reflected our concerns on most health issues and have been responsive and willing to seek our advice on health agendas.

In the fall of 1988 the Coalition held its first annual retreat to create bylaws and operating procedures that would be acceptable to all associations. At the same time, an attempt was made to prioritize long and short term goals. Specific goal categories that would deal with Federal Legislation, State Legislation, Public Education, and Professional Education were developed and a list of issues under each category was considered. Brochure development, communications and election of officers finalized our first annual meeting.

The Coalition deflected many requests during the political season from candidates and from individuals who sought our advice on local issues. But we are committed to only those state and federal issues that affect the health of our citizens.

We are beginning 1989 with a new sense of vigor and support. Our Coalition employes more individuals than any other industry in the State of Alaska. In fact, we rival the state in number of employed, and although we are not a political entity we will strongly pursue any issue that subjugates quality health care for political purposes. However, we will vigorously support any entity that attempts to improve the quality of care for our fellow citizens.

The cost of health insurance today is unaffordable or unavailable for many of us. Yet, the insurance industry, the provider community, and our business leaders continue to level charges that are borne of ignorance and misunderstanding. As this continues, those families caught in the gap are reluctant or unable to seek medical care and the Health Care Coalition of Alaska is concerned.

To address this urgent problem, I will ask the Coalition to sponsor a series of meetings beginning in April that will include Blue Cross, Aetna, and other major health insurance carriers, business leaders both large and small, and representatives from the legislature and governors office. Although I suspect we will never agree on a majority of the issues, we must begin a dialogue of understanding that will consider the public's health first. Self interests must be negotiated and resolved. Our citizens expect the very best from each of us. We must deliver.

Raymond Schalow
Executive Director
Alaska State Medical Association

Auxiliary News

Alaska State Medical Association Auxiliary

The state medical auxiliary was proud to present a total of $3381.05 to the American Medical Society–Emergency Relief Fund (AMA-ERF) Sharing Card. Congratulations to **Patrice Gerster** who organized this successful project!

The state auxiliary is beginning to plan the auxiliary spouse and guest program for the 1989 Alaska State Medical Convention. The convention will be held in Sitka on June 6–11. Sitka is one of the more historically interesting cities in Alaska, just waiting to be explored. A special bonus this year will be performances by musicians from the Sitka Summer Music Festival.

Alice Samuelson
President, ASMA Auxiliary

Anchorage Medical Society Auxiliary

The Anchorage auxiliary will have a general membership meeting with guest speaker, James Patterson, M.D., on February 22, 1989. The meeting, scheduled for 12:30 PM., will be held in the conference room of the ASMA office. Dr. Patterson, chairman of the Impaired Physician Committee, will give us a brief overview of the new "Healing Hands" program. Please plan to attend this meeting.

Jan McCrimmon, Health Projects Chairman, is busy planning the upcoming family workshop for March. This program will be planned with you, the physician family, in mind. Local psychiatrists and therapists will deal with problems specific to physician families as well as other stress of daily life. Detailed invitations will be mailed later in February.

Children's Healthfest '89 will kick off the Anchorage Health Fair Month on April 1, 1989. Because of Healthfest's growing popularity we have been asked to be the first fair of the season. Plans are underway for a wonderful fair with emphasis on health awareness and public safety, and with more emphasis on adolescent health concerns this year. Not only is the Healthfest a great way to provide health awareness and education to the community, but also the high visibility of our physician volunteers helps to improve the physician's image in our communities as well. Please plan to attend the fair, and even better, donate a few hours of your time on April 1, 1989, 10:00 am to 6:00 pm.

Lindsay Hansen Tucker
President, Anchorage Medical Society Auxiliary

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AIR FORCE

Editorial

A new, two-year session of the Alaska Legislature has begun as state revenue projections predict as much as an $800 million shortfall in state revenues. We shivered in the midst of a record cold spell as temperatures have fallen to -68° F and wind chill contributed to effective temperatures of -100° F and worse. My own attention primarily focused on my frozen water pipes and my aging car. After 14 hours, I thawed pipes that, unbelievably, had not burst. And with the help of a strategically placed space heater, a jump start, persistence, and a prayer, my car rewarded my faith in the internal combustion engine, one cylinder at a time. What with the health issues looming ahead for us to tackle, I almost welcomed these challenging diversions.

★ ★ ★ ★

In spite of overwhelming support for our existing immunization laws and our current immunization program, expressed repeatedly by Alaskans in a half-dozen legislative hearings during the last legislative session, we can expect another effort to be mounted to weaken our immunization law. We will again need to expend considerable time and effort to maintain our highly successful immunization program.

★ ★ ★ ★

As the number of individuals with Human Immunodeficiency Virus (HIV) infection and Acquired Immunodeficiency Syndrome (AIDS) continues to mount, so does fear and anxiety. Our knowledge of HIV and AIDS grows constantly, strengthening our scientific understanding of the virus and its transmission. Yet, fear and uncertainty continue to exist. Individuals who harbor unwarranted fears are still afraid. We must face our fears and act in spite of them in order to meet our obligations as physicians and other health care professionals. We face increasing challenges to respond to this tragic epidemic based on the highest standards of scientific evidence and ethical principles.

★ ★ ★ ★

All health care providers need to become familiar with current efforts of the Occupational Safety and Health Administration (OSHA) to become involved in regulating health care workers. OSHA is proposing major expansion of its efforts by becoming involved for the first time with regulation of biologic hazards. Current efforts were initiated about 18 months ago in response to concerns about AIDS, hepatitis B, cytomegalovirus (CMV) and toxoplasmosis. Since then, OSHA appears to have abandoned CMV and toxoplasmosis, focusing instead on AIDS and hepatitis B. The OSHA final proposed standard is due to be published in the Federal Register at any time. If OSHA proceeds with this proposed standard, every heath care worker (including community health aide, fireman, policeman, provider of pre-hospital emergency service, optometrists, podiatrists, physician, nurse, laboratory worker, etc., etc.) and health care facility will be profoundly affected. Every reader of *Alaska Medicine* should take time and expend the effort to become familiar with these OSHA proposals.

★ ★ ★ ★

Legislation has been introduced to the Alaska Senate (Senate Bill No. 77 by Senator Paul Fisher) to require third party notification by health care providers and facilities of all individuals known to be infected with HIV. The bill raises many issues including privacy of the individual, scope and sanctity of the physician-patient relationship, scientific evidence of risk of transmission of HIV, emergency workers' right-to-know, effective uses of public health control measures, and workers compensation. Responding appropriately will require thorough understanding of the scientific evidence about HIV and hepatitis B, and clear perception of the relationship of individual rights, governmental police powers, medical ethics, and social justice.

★ ★ ★ ★

As the articles on tuberculosis in this issue of *Alaska Medicine* document, efforts begun early in the 1950s to control tuberculous have proven to have been more successful than anyone might have dreamed when they were begun. Being, myself, only a relatively recent arrival to Alaska, I am unable to list all of the individuals whose dedicated efforts brought about this dramatic medical and public health triumph. I can, however, express my admiration and gratitude for their work!

★ ★ ★ ★

I certainly was impressed to see how quickly our legislature can get things done. My, no sooner did we find out that making alcoholic beverages in your home was interpreted by the Alcohol Beverage and Control Board to be illegal than a bill was whisked through to make home brewing legal. Now, I wonder if we could get similarly prompt action by the legislature to enact legislation requiring use of seat belts.

* * * *

A major change recently occurred when, under an executive order signed by Governor Steve Cowper, authority for setting Medicaid rates was transferred to the Department of Health and Social Services. The Medicaid Rate Commission, a five member independent body, previously established Medicaid rates. As a result of the recent changes, the commission's recommendations will be advisory with final authority residing with the state health agency. The changes grew from a several year dispute (as yet unsettled) with the Health Care Financing Agency (HCFA) of the federal government over the high costs of the Alaska Medicaid program. Do you suppose that HCFA's economic analysis of the Alaska Medicaid program was of higher quality than the studies of Alaska's health care system that were conducted by the Veteran's Administration?

Letters to the Editor

Dear Editor:

After reading the article by William A. Yasnoff, M.D., Ph.D., on "Electronic Information for Physicians: A New Dimension in Solving Traditional Problems" (AK Med 1988;30 (5):167–168), Dr. Enoch Gordis, Director of the National Institute on Alcohol Abuse and Alcoholism, asked me to contact you. He thought you and your readers might be interested in knowing about two additional sources of electronically available information that are available to physicians and others. For individuals seeking alcohol information, the National Institute on Alcohol Abuse and Alcoholism has developed both a bibliographic resource, the Alcohol and Alcohol Problems Science Database, and a statistical database on alcohol epidemiology called Quick Facts.

The Alcohol and Alcohol Problems Science Database, identified by its label ETOH (after EtOH, one of the chemical designations for ethyl alcohol) is available to all subscribers to BRS Information Technologies, a commercial database vendor. Updated monthly, ETOH provides records and abstracts for over 55,000 published sources on all aspects of alcoholism research including psychology, psychiatry, physiology, epidemiology, sociology, animal studies, treatment, prevention, education, accidents and safety, legislation, employment, labor and industry, and public policy. An online search by subject, author, date or institution can be completed in minutes at costs as low as $16.00 per connect hour and $0.03 per printed abstract. Information on subscriptions to BRS Information Technologies is available at 800-468-0908.

Quick Facts, the electronic bulletin board of the Institute's Division of Biometry and Epidemiology, contains authoritative statistics on alcohol consumption and patterns of use, alcohol-related morbidity and mortality, public attitudes, and other topics that meet epidemiologic research needs of the public health community. New information is added regularly. Users can receive instructions on use of the index, table of contents, and other features when connected to the system. Interested readers may call Quick Facts at 202/289-4112 (modem) or 202/842-7600 (voice).

Terry S. Freeman
Director
Alcohol Research Collection, Analysis,
and Dissemination Project
National Institute on Alcohol Abuse and Alcoholism

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Editor

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Excellence in scientific publication depends on the integrity of the peer-review system. Reviewers have graciously volunteered their time and expertise to assist authorx in improving manuscripts published in *Alaska Medicine. Alaska Medicine* thanks the reviewers for their contributions. —Editor

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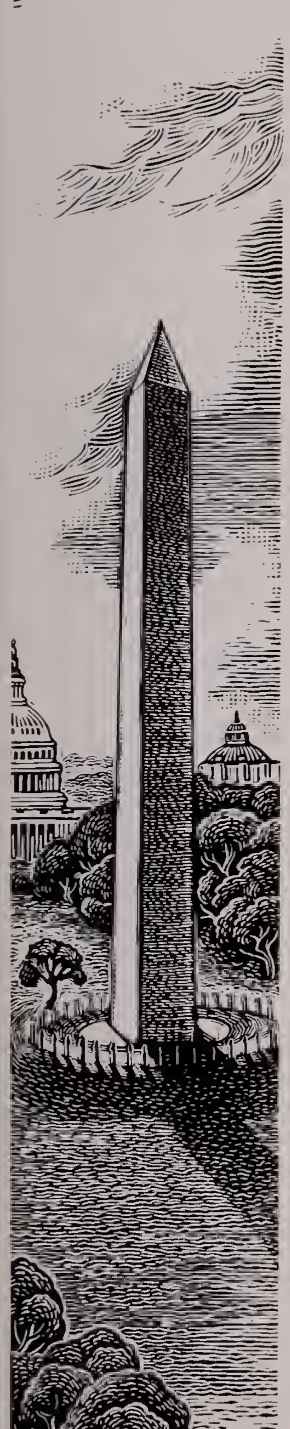

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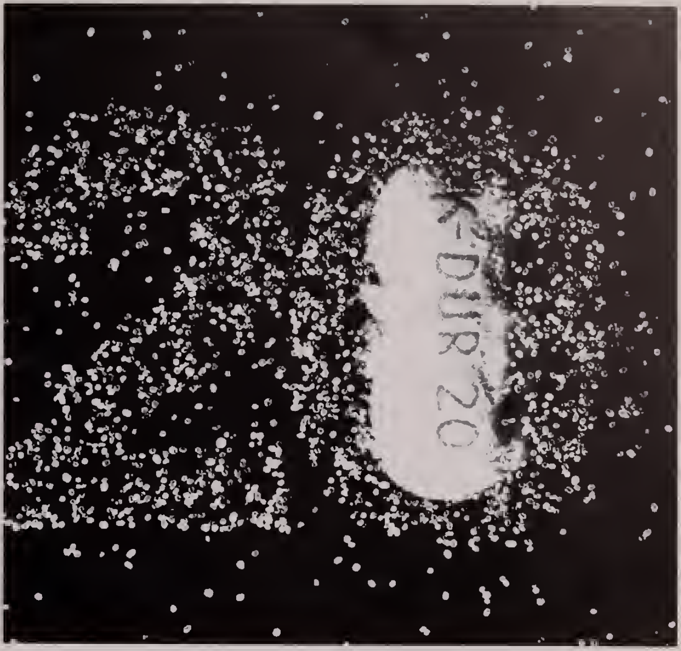

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K-DUR™ Microburst Release System˙ (potassium chloride) Sustained Release Tablets

INDICATIONS AND USAGE: BECAUSE OF REPORTS OF INTESTINAL AND GASTRIC ULCERATION AND BLEEDING WITH SLOW-RELEASE POTASSIUM CHLORIDE PREPARATIONS, THESE DRUGS SHOULD BE RESERVED FOR THOSE PATIENTS WHO CANNOT TOLERATE OR REFUSE TO TAKE LIQUID OR EFFERVESCENT POTASSIUM PREPARATIONS OR FOR PATIENTS IN WHOM THERE IS A PROBLEM OF COMPLIANCE WITH THESE PREPARATIONS

1 For therapeutic use in patients with hypokalemia with or without metabolic alkalosis, in digitalis intoxication and in patients with hypokalemic familial periodic paralysis.

2 For the prevention of potassium depletion when the dietary intake is inadequate in the following conditions Patients receiving digitalis and diuretics for congestive heart failure, hepatic cirrhosis with ascites, states of aldosterone excess with normal renal function, potassium-losing nephropathy, and with certain diarrheal states

3 The use of potassium salts in patients receiving diuretics for uncomplicated essential hypertension is often unnecessary when such patients have a normal dietary pattern. Serum potassium should be checked periodically, however, and if hypokalemia occurs, dietary supplementation with potassium-containing foods may be adequate to control milder cases. In more severe cases supplementation with potassium salts may be indicated

CONTRAINDICATIONS: Potassium supplements are contraindicated in patients with hyperkalemia since a further increase in serum potassium concentration in such patients can produce cardiac arrest Hyperkalemia may complicate any of the following conditions: Chronic renal failure, systemic acidosis such as diabetic acidosis, acute dehydration, extensive tissue breakdown as in severe burns, adrenal insufficiency, or the administration of a potassium-sparing diuretic (e.g , spironolactone, triamterene)

Wax-matrix potassium chloride preparations have produced esophageal ulceration in certain cardiac patients with esophageal compression due to enlarged left atrium.

All solid dosage forms of potassium chloride supplements are contraindicated in any patient in whom there is cause for arrest or delay in tablet passage through the gastrointestinal tract. In these instances, potassium supplementation should be with a liquid preparation.

WARNINGS: Hyperkalemia—In patients with impaired mechanisms for excreting potassium, the administration of potassium salts can produce hyperkalemia and cardiac arrest. This occurs most commonly in patients given potassium by the intravenous route but may also occur in patients given potassium orally Potentially fatal hyperkalemia can develop rapidly and be asymptomatic. The use of potassium salts in patients with chronic renal disease, or any other condition which impairs potassium excretion, requires particularly careful monitoring of the serum potassium concentration and appropriate dosage adjustment

Interaction with Potassium Sparing Diuretics—Hypokalemia should not be treated by the concomitant administration of potassium salts and a potassium-sparing diuretic (e.g., spironolactone or triamterene) since the simultaneous administration of these agents can produce severe hyperkalemia

Gastrointestinal Lesions—Potassium chloride tablets have produced stenotic and/or ulcerative lesions of the small bowel and deaths These lesions are caused by a high localized concentration of potassium ion in the region of a rapidly dissolving tablet, which injures the bowel wall and thereby produces obstruction, hemorrhage or perforation.

K-DUR tablets contain micro-crystalloids which disperse upon disintegration of the tablet. These micro-crystalloids are formulated to provide a controlled release of potassium chloride. The dispersibility of the micro-crystalloids and the controlled release of ions from them are intended to minimize the possibility of a high local concentration near the gastrointestinal mucosa and the ability of the KCl to cause stenosis or ulceration. Other means of accomplishing this (e.g., incorporation of potassium chloride into a wax matrix) have reduced the frequency of such lesions to less than one per 100,000 patient years (compared to 40–50 per 100,000 patient years with enteric-coated potassium chloride) but have not eliminated them The frequency of GI lesions with K-DUR tablets is, at present, unknown. K-DUR tablets should be discontinued immediately and the possibility of bowel obstruction or perforation considered if severe vomiting, abdominal pain, distention, or gastrointestinal bleeding occurs

Metabolic Acidosis—Hypokalemia in patients with metabolic acidosis should be treated with an alkalinizing potassium salt such as potassium bicarbonate, potassium citrate, potassium acetate, or potassium gluconate

PRECAUTIONS: The diagnosis of potassium depletion is ordinarily made by demonstrating hypokalemia in a patient with a clinical history suggesting some cause for potassium depletion In interpreting the serum potassium level, the physician should bear in mind that acute alkalosis per se can produce hypokalemia in the absence of a deficit in total body potassium while acute acidosis per se can increase the serum potassium concentration into the normal range even in the presence of a reduced total body potassium. The treatment of potassium depletion, particularly in the presence of cardiac disease, renal disease, or acidosis requires careful attention to acid-base balance and appropriate monitoring of serum electrolytes, the electrocardiogram, and the clinical status of the patient.

Laboratory Tests: Regular serum potassium determinations are recommended. In addition, during the treatment of potassium depletion, careful attention should be paid to acid-base balance, other serum electrolyte levels, the electrocardiogram, and the clinical status of the patient, particularly in the presence of cardiac disease, renal disease, or acidosis.

Drug Interactions: Potassium-sparing diuretics, see **WARNINGS.**

Carcinogenesis, Mutagenesis, Impairment of Fertility: Long-term carcinogenicity studies in animals have not been performed.

Pregnancy Category C: Animal reproduction studies have not been conducted with K-DUR. It is also not known whether K-DUR can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. K-DUR should be given to a pregnant woman only if clearly needed

Nursing Mothers: The normal potassium ion content of human milk is about 13 mEq per liter. Since oral potassium becomes part of the body potassium pool, so long as body potassium is not excessive, the contribution of potassium chloride supplementation should have little or no effect on the level in human milk.

Pediatric Use: Safety and effectiveness in children have not been established

ADVERSE REACTIONS: One of the most severe adverse effects is hyperkalemia (see **CONTRAINDICATIONS, WARNINGS**, and **OVERDOSAGE**). There have also been reports of upper and lower gastrointestinal conditions including obstruction, bleeding, ulceration, and perforation (see **CONTRAINDICATIONS** and **WARNINGS**); other factors known to be associated with such conditions were present in many of these patients.

The most common adverse reactions to oral potassium salts are nausea, vomiting, abdominal discomfort, and diarrhea These symptoms are due to irritation of the gastrointestinal tract and are best managed by taking the dose with meals or reducing the dose.

Skin rash has been reported rarely

OVERDOSAGE: The administration of oral potassium salts to persons with normal excretory mechanisms for potassium rarely causes serious hyperkalemia However, if excretory mechanisms are impaired or if potassium is administered too rapidly intravenously, potentially fatal hyperkalemia can result (see **CONTRAINDICATIONS** and **WARNINGS**). It is important to recognize that hyperkalemia is usually asymptomatic and may be manifested only by an increased serum potassium concentration and characteristic electrocardiographic changes (peaking of T-waves, loss of P-waves, depression of S-T segment, and prolongation of the QT-interval). Late manifestations include muscle-paralysis and cardiovascular collapse from cardiac arrest.

Treatment measures for hyperkalemia include the following

1 Elimination of foods and medications containing potassium and of potassium-sparing diuretics.

2 Intravenous administration of 300 to 500 ml/hr of 10% dextrose solution containing 10–20 units of insulin per 1,000 ml

3 Correction of acidosis, if present, with intravenous sodium bicarbonate.

4 Use of exchange resins, hemodialysis, or peritoneal dialysis.

In treating hyperkalemia, it should be recalled that in patients who have been stabilized on digitalis, too rapid a lowering of the serum potassium concentration can produce digitalis toxicity

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BRIEF SUMMARY

CONTRAINDICATIONS

There are no known contraindications to the use of sucralfate.

PRECAUTIONS

Duodenal ulcer is a chronic, recurrent disease. While short-term treatment with sucralfate can result in complete healing of the ulcer, a successful course of treatment with sucralfate should not be expected to alter the post-healing frequency or severity of duodenal ulceration.

Drug Interactions: Animal studies have shown that simultaneous administration of CARAFATE (sucralfate) with tetracycline, phenytoin, digoxin, or cimetidine will result in a statistically significant reduction in the bioavailability of these agents. The bioavailability of these agents may be restored simply by separating the administration of these agents from that of CARAFATE by two hours. This interaction appears to be nonsystemic in origin, presumably resulting from these agents being bound by CARAFATE in the gastrointestinal tract. The clinical significance of these animal studies is yet to be defined. However, because of the potential of CARAFATE to alter the absorption of some drugs from the gastrointestinal tract, the separate administration of CARAFATE from that of other agents should be considered when alterations in bioavailability are felt to be critical for concomitantly administered drugs.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Chronic oral toxicity studies of 24 months' duration were conducted in mice and rats at doses up to 1 gm/kg (12 times the human dose). There was no evidence of drug-related tumorigenicity. A reproduction study in rats at doses up to 38 times the human dose did not reveal any indication of fertility impairment. Mutagenicity studies were not conducted.

Pregnancy: Teratogenic effects. Pregnancy Category B. Teratogenicity studies have been performed in mice, rats, and rabbits at doses up to 50 times the human dose and have revealed no evidence of harm to the fetus due to sucralfate. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when sucralfate is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Adverse reactions to sucralfate in clinical trials were minor and only rarely led to discontinuation of the drug. In studies involving over 2,500 patients treated with sucralfate, adverse effects were reported in 121 (4.7%).

Constipation was the most frequent complaint (2.2%). Other adverse effects, reported in no more than one of every 350 patients, were diarrhea, nausea, gastric discomfort, indigestion, dry mouth, rash, pruritus, back pain, dizziness, sleepiness, and vertigo.

OVERDOSAGE

There is no experience in humans with overdosage. Acute oral toxicity studies in animals, however, using doses up to 12 gm/kg body weight, could not find a lethal dose. Risks associated with overdosage should, therefore, be minimal.

DOSAGE AND ADMINISTRATION

The recommended adult oral dosage for duodenal ulcer is 1 gm four times a day on an empty stomach.

Antacids may be prescribed as needed for relief of pain but should not be taken within one-half hour before or after sucralfate.

While healing with sucralfate may occur during the first week or two, treatment should be continued for 4 to 8 weeks unless healing has been demonstrated by x-ray or endoscopic examination.

HOW SUPPLIED

CARAFATE (sucralfate) 1-gm tablets are supplied in bottles of 100 (NDC 0088-1712-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1712-49). Light pink scored oblong tablets are embossed with CARAFATE on one side and 1712 bracketed by C's on the other. Issued 1/87

Reference:

1. Eliakim R, Ophir M, Rachmilewitz D: *J Clin Gastroenterol* 1987;9(4):395-399.

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Please see brief summary of prescribing information, and reference on adjacent page.

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Summary.
Consult the package literature for prescribing information.

Indication: Lower respiratory infections, including pneumonia, caused by *Streptococcus pneumoniae, Haemophilus influenzae*, and *Streptococcus pyogenes* (group A β-hemolytic streptococci).
Contraindication: Known allergy to cephalosporins.
Warnings: CECLOR SHOULD BE ADMINISTERED CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS. PENICILLINS AND CEPHALOSPORINS SHOW PARTIAL CROSS-ALLERGENICITY. POSSIBLE REACTIONS INCLUDE ANAPHYLAXIS.

Administer cautiously to allergic patients.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics. It must be considered in differential diagnosis of antibiotic-associated diarrhea. Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis.
Precautions:
- Discontinue Ceclor in the event of allergic reactions to it.
- Prolonged use may result in overgrowth of nonsusceptible organisms.
- Positive direct Coombs' tests have been reported during treatment with cephalosporins.
- Ceclor should be administered with caution in the presence of markedly impaired renal function. Although dosage adjustments in moderate to severe renal impairment are usually not required, careful clinical observation and laboratory studies should be made.
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
- Safety and effectiveness have not been determined in pregnancy, lactation, and infants less than one month old. Ceclor penetrates mother's milk. Exercise caution in prescribing for these patients.

Adverse Reactions: (percentage of patients)

Therapy-related adverse reactions are uncommon. Those reported include
- Gastrointestinal (mostly diarrhea): 2.5%
- Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment.
- Hypersensitivity reactions (including morbilliform eruptions, pruritus, urticaria, and serum-sickness-like reactions that have included erythema multiforme [rarely, Stevens-Johnson syndrome] and toxic epidermal necrolysis or the above skin manifestations accompanied by arthritis/arthralgia, and frequently, fever): 1.5%, usually subside within a few days after cessation of therapy. Serum-sickness-like reactions have been reported more frequently in children than in adults and have usually occurred during or following a second course of therapy with Ceclor. No serious sequelae have been reported. Antihistamines and corticosteroids appear to enhance resolution of the syndrome.
- Cases of anaphylaxis have been reported, half of which have occurred in patients with a history of penicillin allergy.
- As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely.
- Rarely, reversible hyperactivity, nervousness, insomnia, confusion, hypertonia, dizziness, and somnolence have been reported.
- Other: eosinophilia, 2%, genital pruritus or vaginitis, less than 1%, and, rarely, thrombocytopenia.

Abnormalities in laboratory results of uncertain etiology
- Slight elevations in hepatic enzymes.
- Transient fluctuations in leukocyte count (especially in infants and children).
- Abnormal urinalysis, elevations in BUN or serum creatinine.
- Positive direct Coombs' test.
- False-positive tests for urinary glucose with Benedict's or Fehling's solution and Clinitest® tablets but not with Tes-Tape® (glucose enzymatic test strip, Lilly). [061088L]

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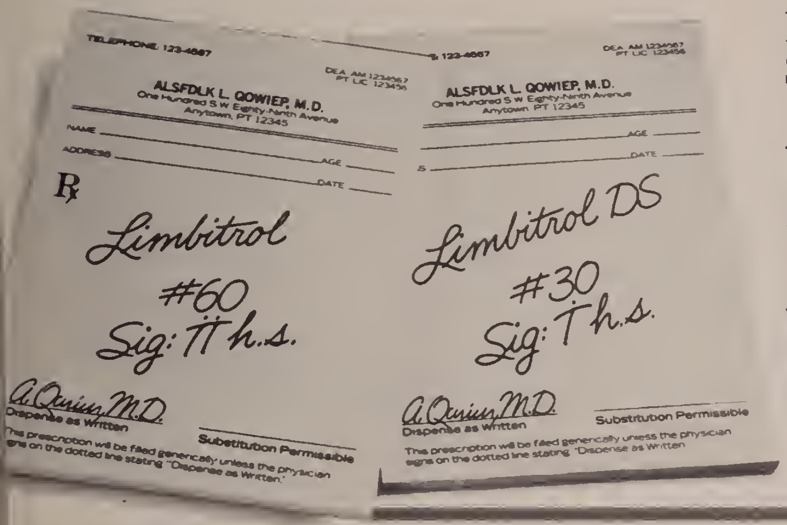

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Limbitrol® DS

Each tablet contains 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) Ⓘⓥ

References: 1. Data on file, Hoffmann-La Roche Inc., Nutley, NJ. **2.** Feighner VP, *et al: Psychopharmacology 61:*217-225, Mar 22, 1979.

Limbitrol® Ⓒ
Tranquilizer—Antidepressant
Before prescribing, please consult complete product information, a summary of which follows:

Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants; concomitant use with MAOIs or within 14 days of monoamine oxidase inhibitors (then initiate cautiously, gradually increasing dosage until optimal response is achieved); during acute recovery phase following myocardial infarction.

Warnings: Use with caution in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur when used with anticholinergics. Closely supervise cardiovascular patients. Arrhythmias, sinus tachycardia, prolongation of conduction time, myocardial infarction and stroke reported with tricyclic antidepressants, especially in high doses. Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations. Consider possibility of pregnancy when instituting therapy.**

Withdrawal symptoms of the barbiturate type have occurred after discontinuation of benzodiazepines (see Drug Abuse and Dependence).

Precautions: Use cautiously in patients with a history of seizures, in hyperthyroid patients, those on thyroid medication, patients with impaired renal or hepatic function. Because of suicidal ideation in depressed patients, do not permit easy access to large quantities of drug. Periodic liver function tests and blood counts recommended during prolonged treatment. Amitriptyline may block action of guanethidine or similar antihypertensives. When tricyclic antidepressants are used concomitantly with cimetidine (Tagamet), clinically significant effects have been reported involving delayed elimination and increasing steady-state concentrations of the tricyclic drugs. Use of Limbitrol with other psychotropic drugs has not been evaluated; sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Should not be taken during the nursing period or by children under 12. In elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

Adverse Reactions: Most frequent: drowsiness, dry mouth, constipation, blurred vision, dizziness, bloating. Less frequent: vivid dreams, impotence, tremor, confusion, nasal congestion. Rare: granulocytopenia, jaundice, hepatic dysfunction. Others: many symptoms associated with depression including anorexia, fatigue, weakness, restlessness, lethargy.
Adverse reactions not reported with Limbitrol but reported with one or both components or closely related drugs: *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke. *Psychiatric:* Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania, increased or decreased libido. *Neurologic:* Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns. *Anticholinergic:* Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue. *Endocrine:* Testicular swelling, gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female, elevation and lowering of blood sugar levels, and syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Other:* Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.

Drug Abuse and Dependence: Withdrawal symptoms similar to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of chlordiazepoxide; more severe seen after excessive doses over extended periods; milder after taking continuously at therapeutic levels for several months. Withdrawal symptoms also reported with abrupt amitriptyline discontinuation. Therefore, after extended therapy, avoid abrupt discontinuation and taper dosage. Carefully supervise addiction-prone individuals because of predisposition to habituation and dependence.

Overdosage: Immediately hospitalize patient. Treat symptomatically and supportively. I.V. administration of 1 to 3 mg physostigmine salicylate may reverse symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.

How Supplied: *Double strength (DS) Tablets,* white, film-coated, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt), and *Tablets,* blue, film-coated, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 50.

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- First-week reduction in somatic symptoms[1]

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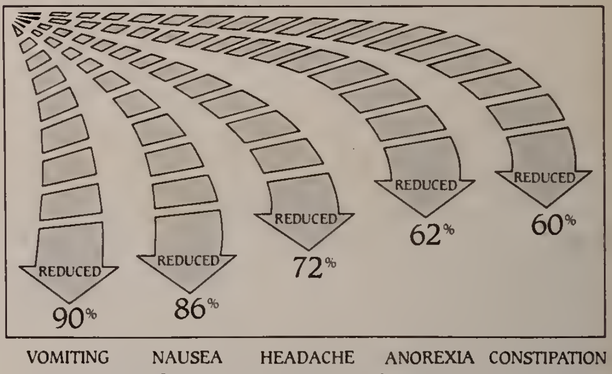
Percentage of Reduction in Individual Somatic Symptoms During First Week of Limbitrol Therapy*



*Patients often presented with more than one somatic symptom.

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Death in Anchorage in 1986

Rodman Wilson, M.D.[1]

ABSTRACT

In 1986, 886 persons died in Anchorage compared to 863 in 1985. The crude death rate was essentially the same in the two years. When age-adjusted, the 1986 Anchorage death rate was higher than in the nation.

From 1985 to 1986, cardiovascular deaths increased, cancer deaths were unchanged, and mortality from violence decreased. However, the rate of violent deaths was still three times the national rate and was especially high among Alaska Natives.

Motor vehicle fatalities were distinctly fewer in 1986 but continued to be strongly linked to alcohol consumption.

Every twenty-first death was from gunfire. Cocaine directly caused eight deaths.

INTRODUCTION

In 1986, 886 persons died in Anchorage. This was 23 more than in 1985. However, the crude death rate was approximately the same in 1986 as in 1985 (Table 1).

Deaths by underlying cause are listed in the Appendix.

Table 1. Crude Death Rate (CDR) for Anchorage 1985 and 1986 Compared to CDR for United States 1986

	Number of Deaths	Estimated Mid-Year Population	CDR per 1,000	Confidence Interval (95%)
Anchorage 1985	863	248,263+	3.48	3.25–3.71
Anchorage 1986	886	248,050+	3.57	3.34–3.80
USA 1986	2,099,000*	241,096,000°	8.71	8.67–8.75

* Provisional, based on sample of 10% of death certificates: Monthly Vital Statistics Report, National Center for Health Statistics 1987:35; No. 13, Aug 24.

+ 1987 Anchorage Population Profile, Department of Community Planning, Municipality of Anchorage.

° U.S. Department of Commerce, Bureau of the Census.

METHODS

I perused and edited death certificates of all persons dying in the Municipality of Anchorage in the same way as I did in 1985(1). I spoke to physicians concerning 151 (17%) of the certificates in order to clarify the underlying cause of death or to decipher abbreviations.

An autopsy was conducted for 305 (34%) of the 886 deaths. Most often it was done in cases of violence or when death occurred out of a hospital and was unexpected. Forensic investigation, almost always including autopsy, occurred in all motor vehicle fatalities, in all suspected cases of drug overdose, suicide, and homicide, and in all instances of unexplained infant death except one.

CORRECTION TO STUDY OF 1985 DEATHS

After reporting 794 deaths in 1985 in Anchorage, I discovered that the Alaska Bureau of Vital Statistics had not provided copies of all 1985 death certificates. In addition, a few duplications and misassignments of year of death surfaced.

The correct number of persons recorded as dying in Anchorage in 1985 is 863. In recalculating rates, Anchorage population was adjusted upward to include persons housed on military bases and individuals living in group quarters such as dormitories and nursing homes.

With these additions and corrections rates and totals change, but conclusions reached in the 1985 survey are not altered because the distribution of errors was random.

In preparing the present report, I personally inspected every 1986 death certificate at the Anchorage registrar's office. Revised numbers for 1985 are presented when comparisons are made between 1985 and 1986.

A copy of the amended tabulation of 1985 deaths is available from the Municipality of Anchorage Department of Health & Human Services.

For purposes of this study, as in 1985, "violent" deaths include "external causes of injury and poisoning" (E800–E999) and alcoholism (among codes 291,303, 425,571,991,E880) of the International Classification of Disease, 9th Revision (ICD–9–CM) 1978. "Violent" and "violence" are not terms used in national classifications of disease based on ICD–9–CM. My use of them is special and is employed to emphasize the frequency of unnatural death in Anchorage and to aver that alcoholism, whether acute or chronic, is essentially a poisoning and is as unnatural as, say, drowning, suicide, or death from auto impact.

Even though records of all registered deaths were collected, numbers are considered to be samples of the "true" population of deaths. Confidence intervals, z and Student's t statistics, and chi-square tests, all at 95% or greater, are used to test statistical significance of rates, means, and other inter-year or subgroup characteristics (2).

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Deaths of persons residing in Anchorage but dying elsewhere were not traced.

RESULTS

Table 2 displays selected characteristics of 1985 and 1986 deaths. A greater proportion of deaths in 1986 were among women than in 1985. Median age at death rose in 1986. Distribution by race and place of residence was little different between the two years. The autopsy rate was lower in 1986 because fewer violent deaths occurred.

Table 2. Selected Characteristics of Deaths 1985 and 1986

	1985		1986		Difference*
	Number	Percent	Number	Percent	
Deaths	863		886		NS
Male	521	(60.4)	492	(55.5)	$p < 0.05$
Female	342	(39.6)	394	(44.5)	$p < 0.05$
Age at Death:					
all cases					
median	58		60		
mean	52.8		53.3		NS
excl age ≤ 1					
median	60		62		
mean	56.7		58.0		NS
excluding death age 1					
Resident of Anchorage	633	(73.3)	652	(73.6)	NS
Race:					
Caucasian$^+$	652	(75.6)	688	(77.7)	NS
Alaska Native	145	(16.8)	141	(15.9)	NS
Black	34	(3.9)	31	(3.5)	NS
Asian	15	(1.7)	8	(0.9)	NS
Other or unknown	17	(1.9)	18	(2.0)	NS
Autopsies	356	(41.3)	305	(34.4)	$p < 0.005$

* Chi-square test: NS (not significant) $p > 0.05$; except mean age difference: NS $z < \pm 1.96$.

+ Including Hispanic.

Table 3 lists the number and proportion of deaths in 1985 and 1986 by broad category of underlying cause. Cardiovascular deaths rose, cancer deaths remained the same, and deaths caused by violence fell in 1986.

There were 53 fewer deaths from violent causes in 1986 than in 1985. While the number of deaths from alcoholism, suicide, homicide, inadvertent drug overdose, and unintentional gunshot were virtually the same in the two years, fatalities due to vehicular collisions were far fewer (36) in 1986 than in 1985 (63). Fire killed 12 in 1985 but only three in 1986. Eight on-the-job fatalities were recorded in 1985 contrasted to three in 1986. Nine drowning or boating deaths occurred in 1985 and only four in 1986. Eight died from falls in 1985 and only one in 1986. However, five children suffocated in 1986 contrasted to one in 1985.

Table 3. Categories of Death in Anchorage 1985 and 1986

	1985		1986	
	Number	Percent	Number	Percent
Cardiovascular disease	241	(27.9)	270	(30.5)
Cancer	208	(24.1)	215	(24.3)
Violence	220	(25.5)	167	(18.8)
Death before age 1	57	(6.6)	70	(7.9)
Lung disease	44	(5.1)	59	(6.6)
Infection	45	(5.2)	36	(4.1)
Neurologic disease	17	(2.0)	28	(3.2)
Gastrointestinal disease	9	(1.0)	18	(2.0)
Other cause	22	(2.6)	23	(2.6)
Total	**863**	**(100.0)**	**886**	**(100.0)**

Twenty-seven (75%) of the 36 fatal motor-vehicle related deaths in 1986 were caused by crashes occurring within Anchorage. The other nine died in Anchorage after collisions elsewhere in Alaska; these nine are not considered further.

Of the 27, 10 died in multi-vehicle collisions, nine in single-vehicle crashes usually involving high-speed rollover, and eight were pedestrians. Sixteen (59%) of the victims were male, 11 female. Median age was 25 years (range 3–75). Fourteen (52%) of the fatal events occurred between 9 P.M. and 7 A.M..

Sixteen (59%) of 27 victims of motor vehicle impacts occurring in Anchorage in 1986 were discovered to have been drinking. Nine had no alcohol in their blood, and two were not tested. Mean blood alcohol among the 16 with detectable alcohol was 210 mg% (median 231, range 17-333). In 1985, 26 (53%) of 49 who died from crashes in Anchorage had been drinking. Twenty had no alcohol in their blood, and three were not tested. Mean blood alcohol among those with measurable levels was lower in 1985—185 mg% (median 190, range 30-381). The difference is not statistically significant (Student's t=0.88, two-tailed, df=40). Taken together, the 42 individuals testing positively for alcohol in the two-year interval had mean blood alcohol concentration of 202 mg% (median 206, range 17-381). Eighteen (43%) of the 42 were driving.

Although vehicular deaths decreased sharply in 1986, fatal vehicle-pedestrian encounters were approximately the same in the two years—nine in 1985 and eight in 1986 including one pedestrian struck by a train in each year. Eleven of the 17 persons on foot were male, six female. Nine were Alaskan Natives. Blood alcohol was measured in 16 of the 17 pedestrians (and are included in the numbers above). Nine were found to have been drinking. Mean blood alcohol for these nine was 240 mg% (median 245, range 60–381).

Eight persons killed in auto accidents in the biennium were found to have cocaine (5) or marijuana (5) or both (2) in forensic samples. Seven of these eight had also been drinking. Five of the eight were drivers. One passenger who died had alcohol, cocaine, and amphetamine in his body.

Altogether cocaine was implicated in 20 deaths during the two-year period (Table 4). It directly caused death of 11 persons—eight unintentional overdoses, one homicide, one undetermined whether homicide or suicide, and one from bacterial endocarditis following intravenous injection. In nine other cases, cocaine was found in individuals dying in auto crash (5), suicide (3), and unintentional gunshot (1), but its role in demise was uncertain.

Gunfire accounted for 42 deaths in Anchorage in 1986. Twenty-four were suicide, 14 homicide, 3 unintentional, and 1 unexplained. Every twenty-first person (4.7%) who died here in 1986 succumbed to gunshot. The proportion was about the same (4.5%) in 1985.

Three-quarters (73.6%) of persons dying in Anchorage in 1986 lived here. One-quarter (26.4%) dwelled elsewhere: 21.2% in other Alaska towns or villages; 5.2% in other states or other countries. Some of these nonresidents were in Anchorage when a fatal disorder struck, while others were brought here gravely ill or injured. Anchorage hospitals are referral centers for much of the state. The city has a large newborn intensive care facility and a sophisticated system for transporting patients by air.

Forty-six (5.2%) of 886 persons dying in Anchorage were from out-of-state. More were male (29) than female (17). Median age was 69 years. Thirty (65%) had been in Alaska for one month or less when they died. Twenty-three (50%) succumbed in June, July, and August. These characteristics suggest that many were summer visitors. Three-quarters (35) died from cardiovascular disorders, cancer, or chronic lung disease, while the remainder fell to other illnesses (4) or to violence (7).

Table 4. Twenty Deaths in Anchorage Involving Use of Cocaine* 1985 and 1986

	1985	1986
Overdose, unintentional	3	5
Suicide:		
Cocaine and alcohol	1	
Cocaine and hanging		1
Cocaine and gunshot		1
Motor vehicle crash:		
Cocaine in body	1	
Cocaine and alcohol in body	3	1
Gunshot, unintentional, while under influence		1
Homicide by injection		1
Injection, undetermined whether homicide, suicide or unintentional		1
Bacterial endocarditis		1
Total	8	12

* But not necessarily the underlying cause of death (see text).

Disproportionately more Alaska Natives than non-Natives perished from violence in both 1985 and 1986. In 1985, 50 (34%) of 145 Alaska Native deaths were classified as violent contrasted to only 24% of persons of all other races combined. In 1986, 38 (27%) of 141 Native deaths were violent compared to 17% for non-Natives. In each year the difference is highly significant (1985: $X^2=7.4$, df=1, $p<0.01$; 1986: $X^2=7.2$, df=1, $p<0.01$).

Fifty-three (60%) of 88 violent deaths among indigenous persons in the two-year interval were known to have been associated with consumption of alcohol, and others probably were. Mechanisms among the 53 included alcoholic liver disease (17), motor vehicle collision (12), acute alcoholism including aspiration while drunk (9), hypothermia (6), ingestion of methanol or insect repellant (3), suicide while intoxicated (2), and assorted other modes (4).

Alaska Natives expiring in Anchorage had half as many deaths attributable to cardiovascular disease as non-Natives in the 1985–1986 interval. This is consistent with the Natives' younger median age at death (53 years) compared to non-natives (59 years). Alaska Natives also had less cancer, but the difference does not reach statistical significance ($X^2=2.8$, df=1, $p<0.05$). However, five of the seven persons dying from carcinoma of the gallbladder or bile ducts in the two-year period were Natives.

DISCUSSION

As in 1985, the 1986 Anchorage crude death rate was less than half the rate in the United States (Table 1). But after age-adjusting by the "direct" method, using the 1940 U.S. Census as the standard population, Anchorage rates in both years were actually higher than in the nation (Table 5). Anchorage rates were 14% above the national rate in 1985 and 17% higher in 1986. It should be kept in mind that adjusted rates are contrived numbers. Although they do not represent actual experience, they do correct for dissimilar characteristics in samples or populations—in this instance the youthfulness of Anchorage people.

Rates must be interpreted with great caution, as pointed out in the 1985 study (1). Because Anchorage is a referral center, the number of deaths swells without increasing the city's population. This inflates the death rate. The "true" rate cannot be figured because the denominator—the population in which the deaths occur—is imprecise. Even if deaths only among residents were counted, including tracing out-of-town deaths, one could not be fully confident about the rate, for the

Table 5. Crude Death Rate (CDR) and Age-Adjusted Death Rate (AADR) per 1000 Anchorage and United States 1985 and 1986

	CDR	AARD*
Anchorage		
1985	3.48	6.20
1986	3.57	6.34
USA		
1985	8.74	5.46
1986	8.71+	5.40

* By direct method (see text).

+ Provisional, based on 10% sample.

Sources for USA CDR and AADR: Monthly Vital Statistics Report, National Center for Health Statistics 1987:35;No.13, Aug 24 and 1987: 36;No.5 (Suppl), Aug 28.

numerator is also suspect: who is a resident? Addresses listed on death certificates were accepted as correct, but it is apparent that many villagers gave a local address upon arrival in Anchorage. For these and other reasons death rates must be read circumspectly.

More Anchorage residents were male (51.9%) than female (48.1%) in 1985 in contrast to the nation where 48.7% of persons were male in 1985 (3); and far more men than women died in the city in both 1985 and 1986. However, the proportion of female deaths increased between the two years from 39.6% to 44.5%.

The population of Anchorage remains young: for 1985, the estimated median age was 27.2 years compared to 31.7 years nationally (3). However, the proportion of persons who are elderly has been rising in recent years. In 1980, only 2.2% of individuals in the city were 65 years of age or older. By 1985, this proportion had risen to 2.8% — a 27% increase, representing an estimated 3,059 more elderly persons. Data for 1986 are not available. The percentage of persons in Anchorage 65 years of age or older is still far below that in the country as a whole. In 1985, 12% of the population of the nation was at least 65 years of age (3).

A change in age distribution will profoundly alter both patterns of death and mean age at death. There was a suggestion of the latter in 1986; mean age at death (excluding death before age 1) rose from 56.7 years in 1985 to 58.0 years in 1986, but the difference is not statistically significant.

Although the number of violent deaths fell in 1986, the unadjusted rate was still approximately three times the national rate. Violence caused 18.8% of deaths in 1986 compared to 25.5% in 1985. Many fewer fatal vehicle crashes occurred in 1986—36 contrasted to 63 in 1985. This alone accounts for half the difference in violent deaths between the two years. In addition, there were fewer drownings, fire deaths, on-the-job lethal injuries, and fatalities from falls at home, though more children were asphyxiated in 1986.

Why fewer fatal auto crashes occurred is unknown. There may have been less auto use because of declining economic activity. Police focused more attention on traffic enforcement in 1986. Safety belt use may have increased. Also, it is possible that Mothers Against Drunk Driving (MADD) may have changed driving habits. Non-alcoholic nightclubs may have kept a few young people from cruising about drunk. Traffic fatalities continued preeminently to involve unbuckled youth, alcohol, drugs, high speed, and darkness.

CONCLUSION

Age, race, gender, migration, patient referral patterns, misuse of drugs, alcohol, firearms and motor vehicles, abuse and neglect of persons, suicide, homicide—all these factors and many others influence incidence of disease and death in a community. It behooves sociologists, planners, teachers, physicians, political leaders, and citizens to keep abreast of these shifting phenomena in order to understand our culture and shape it felicitously.

ACKNOWLEDGMENTS

Kim Armstrong and Chris Curry prepared the data base. Rose Courtright typed the manuscript.

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APPENDIX

Underlying Cause of Death of 886 Persons Dying in Anchorage in 1986

(code numbers are from ICD-9)

CARDIOVASCULAR DISEASE				**270 (30.5%)**
Heart Disease*			**186**	
coronary artery (410,414)		163		
acute myocardial infarction (410)	50			
cardiomyopathy, idiopathic (425.4)		7		
hypertensive (402,404)		6		
rheumatic (393–398)		6		
aortic valvular stenosis, idiopathic (424.1)		2		
congenital atrioventricular canal defect and patent ductus (745.69)		1		
type undetermined (429.9)		1		

***excluding infections of the heart, alcoholic cardiomyopathy, and death from heart disease before age 1.**

Vascular Disease			**84**	
cerebral thrombosis (434.0)		48		
cerebral hemorrage*		9		
aneurysm, Circle of Willis (430)	4			
intraparenchymal (431)	3			
arteriovenous malformation (747.81)	2			
pulmonary embolism (415.1)		7		
atherosclerosis (440)	7			
aortic aneurysm, abdominal (441.3, 441.4)		6		
vascular insufficiency of intestine (557)		3		
cerebral embolism (434.1)		2		
atherosclerosis of carotid artery (433.1)		1		
femoral artery aneurysm (442.3)		1		

***excluding neonatal cerebral hemorrhage**

CANCER				**215 (24.3%)**
Lung (162)		66		
Breast (174)		25		
Colo-rectal (153, 154)		15		
Brain (191)		14		
Metastatic, origin undetermined (199.0)		10		
Pancreas (157)		8		
Esophagus (150)		7		
Stomach (151)		7		
Kidney (189)		7		
Prostate (185)		7		
Malignant melanoma (172)		6		
Ovary (183)		5		
Gallbladder (156.0)		4		
Myeloid leukemia (205)		4		
Lymphoid leukemia (204)		3		
Lymphoma (200–202)		3		
Bladder (188)		3		
Cervix (180)		3		
Endometrium (182)		2		
Myeloma (203)		2		
Oral (140–149)		2		
Larynx (161)		2		
Liver, primary (155.0)		2		

Mastoid (160.1)		1		
Parotid (142.0)		1		
Adrenal (194.0)		1		
Gastrinoma (157.4)		1		
Duodenum (152.0)		1		
Anus (154.3)		1		
Embryonal (186)		1		
Sarcoma of bone, Ewing's (170)		1		
VIOLENCE AND ADVERSE EFFECTS				**167 (18.8%)**
Alcoholism			**40**	
alcoholic liver disease (571)		18		
acute alcoholism (303.0)		6		
chronic alcoholism (303.9)		5		
hypothermia (991.6)		5		
alcoholic cardiomyopathy (425.5)		3		
fall, head injury while intoxicated (E888)		2		
delerium tremens (291.0)		1		
Vehicular Accidents			**36**	
automobile (E812)		24		
auto–pedestrian (E814)		8		
motorcycle (E812.2)		1		
auto–sled (E813)		1		
all–terrain vehicle (E821)		1		
train–man lying on tracks drunk (E805.2)		1		
Suicide			**38**	
gunshot (E955)		24		
motor vehicle exhaust (E952.0)		5		
hanging (E953.0)		4		
drug		3		
amitriptyline (E950.3)	2			
diazepam (E950.3)	1			
drowning (E954)		1		
jumping (E957)		1		
Homicide			**18**	
gunshot (E965)		14		
stabbing (E966)		2		
bludgeoning (E968.2)		1		
cocaine injection (E962.0)		1		
Poisoning, unintentional			**8**	
cocaine (E855.2)		5		
methanol (E860.2)		1		
tricyclic antidepressant (E854.0)		1		
fluorinated hydrocarbon aerosol (E869.8)		1		
Asphyxia, unintentional (E913)			**5**	
age, 13 da, smothered sleeping with adult		1		
2 mo, caught between couch back and cushion		1		
13 mo, dress caught on toy in crib		1		
2 yr, strangled in venetian blinds		1		
5 yr, strangled on jungle gym		1		
Undetermined whether accident, suicide, or homicide			**5**	
gunshot (E895)		1		
blow to chest (E988.8)		1		
motor vehicle exhaust (E982.0)		1		
cocaine (E980)		1		
phenytoin ingestion (E980.4)		1		
Drowning (E910)			**4**	
Fire (E890)			**3**	

Crushing			3	
jack slipped under auto (E916)		1		
concrete slab fell on–job (E916)		1		
cave–in ditch on–job (E913.3)		1		
Miscellaneous			7	
gunshot, accidental (E922)		3		
blow to head, accidental on–job (852.3)		1		
fall, fracture of hip (820)		1		
explosion in motor home (E825)		1		
injury, type undetermined (skeletal remains) (E988.9)		1		

DEATH BEFORE AGE 1*				**70 (7.9%)**
Immaturity (765)		31		
Congenital anomalies (740–759)		12		
Sudden infant death syndrome (798.0)		10		
Birth asphyxia (768)		3		
Aspiration of meconium (770.1)		2		
Fetal hydrops (778.0)		2		
Neonatal intracranial hemorrhage (431)		2		
Congenital heart failure (779.8)		1		
Congenital pneumonia (770.0)		1		
Hemophilus b meningitis (320.0)		1		
Meningococcemia (036.3)		1		
Reye's syndrome (331.81)		1		
Viral myocarditis (422)		1		
Volvulus around Meckel's diverticulum (560.2)		1		
Connective tissue tumor, perineal, benign (215.6)		1		

***except unintentional asphyxia**

LUNG DISEASE*				**59 (6.6%)**
Chronic obstructive pulmonary disease (496)+		43*		
Asthma (493)		4		
Bronchopulmonary dysplasia (770.7)		2		
Pulmonary fibrosis, cause undetermined (516.3)		2		
Bronchiectasis (494)		1		
Tuberculosis (011)		1		
Rheumatoid lung disease (714.81)		1		
Lung disease due to kyphoscoliosis (737.3)		1		
Hypoventilation due to obesity (278)		1		
Asbestosis (501)		1		
Byssinosis (504)		1		
Silicosis (502)		1		

***excluding pneumonia**
+COPD was listed on 22 other certificates as a significant associated condition

INFECTION*				**36 (4.1%)**
Pneumonia (480–486)		17		
Acquired immunodeficiency syndrome (042)		7		
Septicemia (038)		3		
Hepatitis B (070.2)		3		
Meningitis		3		
pneumococcal (320.1)	2			
hemophilus b (320.0)	1			
Acute viral pneumonia & myocarditis (422)		1		
Acute bacterial endocarditis, staphylococcal (421.0)		1		
Chronic pyelonephritis (590.0)		1		

***not including death from infection before age 1**

NEUROLOGIC DISEASE		**28 (3.2%)**
Senile dementia, Alzheimer's disease, organic brain disease (290.0, 330, 331)	14	
Parkinsonism (332)	3	
Amyotropic lateral sclerosis (335.20)	2	
Epilepsy (345)	2	
Multiple sclerosis (340)	1	
Huntington's chorea (333.4)	1	
Progressive bulbar palsy (335.22)	1	
Duchenne progressive muscular dystrophy (359.1)	1	
Metachromatic leukodystrophy (330.0)	1	
Pena-Shokier syndrome (759.8)	1	
Toxic encephalitis, cause undetermined (349.82)	1	
GASTROINTESTINAL		**18 (2.0%)**
Acute pancreatitis, non-alcoholic (577.0)	2	
Acute appendicitis (540)	2	
Volvulus of intestine (560.2)	2	
Bleeding gastric ulcer (531.0)	1	
Perforated gastric ulcer (531.1)	1	
Perforated duodenal ulcer (532.5)	1	
Perforated small bowel, cause undetermined (569.83)	1	
Hemorrhagic gastritis (535.5)	1	
Gastrointestinal hemorrhage, cause undetermined (578.9)	1	
Acute ulcerative colitis (556)	1	
Cirrhosis of liver, non-alcoholic (571.5)	1	
Acute cholecystitis (575.0)	1	
Common duct stones with cholangitis (574.5)	1	
Incarcerated ventral hernia (552.2)	1	
Incarcerated inguinal hernia (550.1)	1	
MISCELLANEOUS		**23 (2.6%)**
Aspiration of gastric contents, not while intoxicated (934.9)	3	
Diabetes mellitus*, not including ESRD (250)	3	
Diabetic end-stage renal disease (250.4)	3	
Chronic renal failure, cause unstated (585)	3	
Chronic glomerulonephritis (582)	1	
Polycystic renal disease, end-state (753.1)	1	
Hemolytic uremic syndrome (283.1)	1	
Panhypopituitarism (253.2)	1	
Sickle cell anemia (282.6)	1	
Rheumatoid arthritis of spine with quadriplegia (720.0)	1	
Hepatic toxicity due to carbamazipine (E936)	1	
Bone marrow hypoplasia due to colchicine (E944.7)	1	
Inanition (263)	1	
Senility (797)	1	
Undetermined natural cause (798.9)	1	
TOTAL		**886 (100.0%)**

***Diabetes mellitus was mentioned a total of 45 times (5.1%) among 886 deaths.**

Thrombotic Thrombocytopenic Purpura

by Richard L. Neubauer, M.D.[1]

Case Report

HISTORY

A 40-year old nursing instructor was admitted to the hospital with severe headache, hematuria and proteinuria, and thrombocytopenia on May 3, 1988.

The patient became ill two weeks prior to admission with severe global headaches. These were attributed to eye strain. She took up to six aspirin a day without benefit. She also described a "rip" in her visual field which was apparent in both eyes and resolved spontaneously over a few hours.

For four to five days before admission, she became increasingly lethargic and developed ecchymoses on her legs and arms. She also noted dark urine. She denied fever or chills. To a friend, she didn't seem herself, and there were subtle personality changes apparent.

About two weeks before admission, she had a brief episode of diarrhea which resolved. Otherwise she had been well. There was no known toxic or drug exposure. She had no recent travel experiences.

The day prior to admission, a CBC was ordered which showed a hemoglobin of 10.7, hematocrit of 29, and platelet count of 26,000.

Past medical history was uneventful.

PHYSICAL EXAMINATION

On examination, she was mildly jaundiced and appeared ill and somewhat inattentive. Vital signs were normal, and she was afebrile. Ophthalmologic examination was normal, including flat optic discs. Her sclerae were slightly icteric. There was no lymphadenopathy. Cardiopulmonary examination was normal. Abdominal examination was normal and without organomegaly. Extremity examination showed numerous ecchymoses on her arms and legs which were non-raised and non-tender. Neurologic examination showed no focal deficits, and she was oriented but lethargic and seemed detached.

LABORATORY VALUES

CBC showed a hemoglobin of 9.8, hematocrit of 27.8, and white cell count of 6,300. Differential included 28% polys, 10% lymphs, 62% monos, and platelet count was 11,000. A peripheral smear showed schistocytes consistent with intravascular hemolysis. A bone marrow aspirate and biopsy showed many megakaryocytes, and left shifted red and white cell precursors, as well as depleted iron stores. Rheumatoid factor, anti-DNA titer, and ANA were all negative. A chemistry panel showed a total bilirubin of 3.0, a direct bilirubin of 0.4, a normal SGOT and SGPT, but the LDH was 698 U/L (nl 100-190 U/L).

Coagulation studies included a prothrombin time of 12.4/11.3 control, partial thromboplastin time 21.8/27.1 control, fibrinogen was 334 mg/dl, and fibrin split products were present in a significant titer. Urinalysis showed 1+ protein, 10-15 RBC/HPF, but was otherwise normal. Both direct and indirect Coombs' tests were negative. A chest x-ray was normal. Blood urea nitrogen was 14, and serum creatinine was 0.7.

SUBSEQUENT COURSE

A diagnosis of Thrombotic Thrombocytopenic Purpura (TTP) was felt to be secure on clinical and laboratory grounds. An overnight trial of fresh frozen plasma (FFP) infusion was begun. However, the next morning her blood counts had worsened. The hematocrit was 21.9, with a platelet count of 13,000. Her headache remained severe. Plasmapheresis was begun, using daily 3-liter exchanges over the following six days. Also, aspirin 325 mg daily and dipyridamole 50 mg three times a day were added.

Her blood counts immediately begun to improve. One week following admission, the hematocrit was 34 and platelet count 133,000. The LDH normalized, and her headaches dramatically improved, although they did not completely resolve. The only complications of the plasmapheresis were transient symptoms during apheresis suggesting hypocalcemia (attributed to possible citrate toxicity), and these were treated successfully with calcium infusion.

She had several episodes of transient visual disturbance similar to those that preceded the admission, but an opthalmologist found no intraocular pathology. An EEG was also normal.

She was discharged, and platelet count and CBC were monitored daily. At first the platelet count continued to rise to 204,000. Her hemoglobin was also rising on a daily basis. However, four days after plasmapheresis was stopped, the platelet count fell to 120,000 and 2 mg of vincristine was administered. The next day the platelet count had fallen further to 46,000 along with a mild drop in the hemoglobin, and she was readmitted to the hospital.

On admission, schistocytes were again seen on peripheral smear. The LDH had risen to 312 U/L, total bilirubin was again elevated at 1.7, and clotting studies were similar to the first admission. Her BUN and creatinine remained normal.

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Daily plasmapheresis was reinitiated, but the volume of each exchange was increased to four liters. Solu–Medrol was begun at a dose of 20 mg intravenously every six hours, and aspirin and dipyridamole were continued. A total of seven plasma exchanges were done, and the platelet count increased to 199,000. A second dose of vincristine was administered about one week after the first dose.

All the other laboratory values also normalized. During this second hospitalization, she displayed febrile episodes without apparent infectious etiology. She also had atypical chest pains and her electrocardiogram showed nonspecific ST and T wave changes. Serial cardiac enzymes were not indicative of myocardial damage.

She was again discharged from the hospital, after surgical placement of a tunneled dialysis catheter. Her platelet count continued to rise and four days after discharge was 304,000. She was still feeling somewhat lethargic and had a mild residual headache, but was otherwise feeling markedly improved. The corticosteroids were tapered quickly, and over almost a year the platelet count and other laboratory and clinical parameters have been indicative of a sustained remission. She will continue to be monitored for possible relapses.

Discussion

INTRODUCTION

Thrombotic Thrombocytopenic Purpura (TTP) was first described by Moschowitz in 1925 (1). The syndrome is characterized by a pentad of symptoms including fever, hemolytic anemia, thrombocytopenia, neurologic signs, and renal involvement. The mortality rate of TTP was 80% or greater until the last two decades when whole blood exchange transfusions, plasma infusions, and most recently, plasmapheresis, were introduced. With these techniques, survival rates have dramatically improved, and 80% survival is now to be expected. Other treatment modalities include anti–platelet agents, corticosteroids, vincristine, and splenectomy.

The etiology of the disorder remains unclear in most cases, but the dramatic success of plasmapheresis provides some insight into the underlying pathogenetic mechanism(s). At least a subset of patients with TTP may have a platelet aggregating factor (PAF). Others may be deficient in a plasma factor, possibly a prostacyclin precursor. A more general term, thrombotic microangiopathy, has been coined that includes other related disorders including hemolytic uremic syndrome (HUS), TTP related to cancer and anti–cancer drugs, and TTP related to pregnancy.

In some of these disorders, the underlying mechanism of the disease is better defined than in TTP. HUS and TTP may be on the opposite ends of a disease spectrum, but there is considerable overlap between the two diseases (2).

CLINICAL MANIFESTATIONS

The three major events clinically in TTP are hemolysis, thrombocytopenia, and altered organ function secondary to the vascular lesions characteristic of TTP. The pentad of symptoms characteristic of TTP has already been outlined. Of these, the three most constant features are hemolytic anemia, thrombocytopenia, and the neurologic symptoms (3). Fever, and renal involvement are more variable. Differential diagnostic considerations in TTP include Evans syndrome (Idiopathic Thrombocytopenic Purpura and Coombs' positive hemolytic anemia), DIC, and Lupus with an associated thrombotic microangiopathy.

The hemolytic anemia is manifested by schistocytes on peripheral smear, hyperbilirubinemia (mostly indirect bilirubin fraction), and elevation of the serum LDH. The thrombocytopenia may be quite severe, and purpuric lesions are common, but gross hemorrhage is rare. The marrow may show megaloblastic changes reflecting folate deficiency from rapid turnover and should show adequate megakaryocytes.

Organ system dysfunction is most commonly overt in the central nervous system. The neurologic symptoms may fluctuate rapidly and may include alterations in mentation, headaches, blurred vision, and seizures (4). Visual symptoms may be related to retinal detachment, vitreous hemorrhage, or they may be central in origin. Renal manifestations include microscopic hematuria, proteinuria, or azotemia. The vascular lesions of TTP are widely distributed. For instance, cardiac involvement on autopsy studies is very common. Clinically, however, cardiac symptoms are usually either inapparent or subtle.

The duration of the illness is less than 90 days in 80% of patients, and less than one year in another 14%. A chronic relapsing form of TTP is described and occurs in a minority of affected patients.

PATHOPHYSIOLOGY

The intraluminal microthrombi in TTP are described as hyaline material deposited primarily in the arterioles and capillaries, and composed of platelets, fibrin, and von Willibrand factor (vWF) by immunofluorescent studies (5). The predominant component of the lesions is, however, platelets. There is also swelling and proliferation of the endothelium of the involved vessel.

The hemolytic anemia is felt to be secondary to contact of circulating red cells with the microvascular thrombi just described.

Fibrin degradation products (FDP's) may be elevated, but fibrinogen turnover rate is normal or near normal (6). Disseminated intravascular coagulation (DIC) is not felt to be directly involved in pathogenesis.

There may, however, be defects in the fibrinolytic system associated with the vascular lesions.

ETIOLOGY

The etiology of TTP is not fully elucidated. The pathways by which the syndrome occurs may be multiple and include:

a) platelet aggregating protein(s)
b) endothelial damage (7)
c) decreased prostacyclin (PGI2) production.

TTP, or more generally, thrombotic microangiopathy (TM), has been observed in association with a variety of infections. Prominent among these is an association between TM and hemorrhagic colitis caused by strains of *E. coli,* and specifically *E. coli* 0157:H7 (8). These strains produce an endotoxin (verotoxin) which causes platelet aggregation and may lead to the syndrome (9). A probably identical toxin is produced by *Shigella dysenteriae* which can also be associated with TM.HIV infection has been described in association with hemolytic uremic syndrome (10). The HIV infected patient described had multiple risk factors including an immune deficiency syndrome, infection, multiple drug therapy, and neoplasia (Kaposi's sarcoma).

Secondary forms of TM are also found in conjunction with malignancy, especially gastric, breast and lung carcinoma (11). Tumor micro-emboli and fibrin thrombi have been found in this syndrome, and platelet consumption may be related to contact with the damaged microvasculature. As in "idiopathic" TTP, DIC is not felt to be a precipitating factor.

TM may also be associated with cancer chemotherapy, especially with regimens containing mitomycin C. Usually this causes a picture similar to HUS with prominent renal involvement. A peculiarity of this particular syndrome is that the administration of red cells of any kind, even washed red cells, may exacerbate the condition, and are to be avoided if at all possible.

Pregnancy can be associated with a variety of syndromes with TM as a feature. The major distinction to be made in pregnancy is between TTP and TM associated with eclampsia/pre-eclampsia. This is important since delivery is key to treatment of eclampsia/pre-eclampsia whereas it will not alter the course of TTP (12).

The association between lupus erythematosus and TM is apparently not a strong one. The presence of vasculitic lesions in lupus may be of importance in cases showing signs of TM.

The observation of a defect in PGI2 availability in some cases may be of pathogenetic importance (13). If this is the case, plasmapheresis may work by supplying a deficient precursor for synthesis of this substance, an important endogenous inhibitor of platelet aggregation.

A platelet agglutinating protein (PAP p37) has been observed and characterized from the serum of a TTP patient (14-16). This protein has been found in the sera of three out of five patients with TTP that were tested. Platelet agglutination caused by PAP p37 was inhibited by IgG from normal adults and from TTP patients after recovery, but not by anti-platelet drugs. The investigators hypothesize that the PAP p37 is a foreign substance, possibly related to an unknown infectious agent.

Finally, a variety of other observations have been made, the significance of which is not known. Large vWF multimers may be present, especially in patients with chronic relapsing TTP. Immune complexes may be present, but are generally not felt to be directly linked to pathogenesis. Primary immune vascular injury has been postulated, but evidence to support such a mechanism is lacking.

TREATMENT

As recently as ten years ago, TTP was treated primarily with anti-platelet agents, corticosteroids, and splenectomy (17,18). The results were variable, generally poor, and probably worse than reflected in the literature due to under reporting of poor results.

Plasma infusion (19) and plasmapheresis (20) were introduced in the 1970s and have had a dramatic effect on improved survival in TTP (21,22). Preceding plasma infusion and plasmapheresis, fresh whole blood exchange transfusions were utilized with some success in the late 1950s. This led to an improvement from a fatality rate of 80% or more to remissions in about 50% of patients.

The use of plasma infusions alone rather than whole blood exchange transfusions was apparently suggested by the concept that plasma of TTP patients may have a deficiency that leads to vasculopathy and/or intravascular platelet aggregation. The response rate with the use of plasma infusion is around 60%. The amount of plasma needed is not well defined, but the volumes used may place the patient at risk for volume overload.

Plasmapheresis along with fresh frozen plasma (FFP) replacement was tried in the 1970s and has since become the most uniformly successful treatment modality for TTP. The volumes of plasma removed and replaced are larger than can be accommodated with plasma infusion alone. The procedure may work by either removal of an inciting agent such as an immune complex, cytotoxic anti-endothelial antibodies, or a platelet aggregating factor, or by supplying a missing factor such as PGI2 precursors. Whatever the mechanism of action, overall response in regimens including plasmapheresis seems to be around 80%.

During treatment, as the platelet count begins to increase, there is a theoretical, and possibly real, concern that the increasing platelet count can supply the "fuel" for increased intravascular platelet aggregation that may lead to clinical worsening of the TTP syndrome

(23). This has led to a recommendation that anti-platelet agents be started as the platelet begins to rise. The same concern has been voiced in regards to platelet infusions during the acute phase of the illness, and it may be best to avoid giving platelets during the acute phase of the illness. There is not total agreement on this point, however (24).

As mentioned, corticosteroids, anti-platelet agents and splenectomy have been employed in TTP since before the above modalities. Their exact role in treatment remains somewhat unclear since large scale randomized trials in this illness have not been practical. Due to the rarity and high mortality rate of the illness, patients with TTP have generally been treated with multiple agents. Recently vincristine has been used with some successes noted (25). The rationale for the use of this drug is unclear. Splenectomy is reserved for severe refractory cases as a last resort.

From the above considerations, a treatment protocol has emerged for TTP. First, prompt diagnosis is helpful and may even account for some of the improved survival noted in recent years. When the diagnosis is made, therapy is begun with infusion of fresh frozen plasma, or whole blood exchange transfusions. If that is unsuccessful, plasmapheresis is begun with 3 liter plasma exchanges. If the initial presentation is very severe, then plasmapheresis may be started immediately. The volume of each plasma exchange may be increased if the response is unsatisfactory (26). Fresh frozen plasma is used to replace the removed plasma, and daily apheresis should be continued until the platelet count normalizes. Anti-platelet agents may be added as the platelet count rises (27). Corticosteroids and vincristine may be added if plasmapheresis alone is not satisfactory. The platelet count, hemoglobin and hematocrit, and serum LDH may be followed as well as clinical symptoms to gauge response.

Plasmapheresis has often been regarded as a very safe procedure, but it is not risk free (28). Prominent among the risks are complications of venous access including infection which may be severe or fatal. Citrate toxicity may occur, especially in patients with liver disease. Rapid fluctuation of serum drug levels may occur as a result of plasma exchange. Finally, exposure of the patient to hepatitis virus or HIV infection depend on the type of replacement fluid being used.

SUMMARY

This case represents a classical presentation of severe TTP. At the time of diagnosis, the patient had a markedly depressed platelet count and evidence of severe hemolytic enemia. Her neurologic symptoms were not ominous at the time of initial presentation but may have worsened rapidly without treatment.

She also demonstrates the success of current treatment modalities. A combination of plasma infusion followed by plasmapheresis, anti-platelet agents, corticosteroids, and vincristine was employed. As in most cases of TTP reviewed in the medical literature, it is difficult to sort out which treatment modality was most responsible for the improvement. Since this is such a deadly disease, there is a tendency to treat with a combination of agents in addition to plasmapheresis. Nonetheless, an impression remains that plasmapheresis was most closely associated with her recovery.

As an additional comment, the use of plasmapheresis in this patient represented the first use of a pheresis machine that had been obtained in Anchorage, Alaska at the urging of the medical community. Our location, remote from academic centers, has made the use of technology such as plasmapheresis less accessible. The presence of plasmapheresis capabilities in a community setting such as ours raises many questions pertaining to the appropriateness of use of the technology, management of complications, etc.. We plan to carefully monitor these parameters.

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Psychiatric Consultation To The Bush

by Paul Kettl, M.D.[1]

Psychiatry in America's second youngest state is still a bit young, and many of the problems in the delivery of good psychiatric care remain a challenge. Over the past two decades psychiatric care in Alaska has traversed a great way—with the establishment of the Alaska Psychiatric Institute, private inpatient psychiatric and substance abuse facilities, as well as the establishment and strengthening of the community mental health system. Anchorage, and to some extent, Fairbanks, are at the forefront of modern psychiatric care delivery, offering their citizens specialized care in virtually all areas of psychiatry through a variety of private and public care networks.

However, the rest of Alaska is not as fortunate; delivering psychiatric care to the bush is a problem that continues. While half of the state's population is centered in Anchorage, and another substantial fraction is located in Fairbanks, the rest of the population is spread over an area one-fifth the size of the continental United States. Population centers ring the coast, but these centers, the metropolitan hub of their own areas, have only a few thousand people—not enough to support a psychiatrist. From these hubs string a series of villages and smaller towns widely dispersed over great areas.

There are many unavoidable problems confronting the development of a comprehensive mental health care system for "the bush." Nowhere else in the country has the problem of trying to bring mental health care to such a dispersed area, with transportation connecting places only by air, and then only when the weather is good. Delivering care to each village presents a number of difficult obstacles. Transportation costs, either in transporting patients from the bush or medical specialists to the area, can be overwhelming, increasing the cost of an already costly system.

Another unavoidable problem facing psychiatry in Alaska is the stigma surrounding psychiatric disorders and psychiatric patients. Fortunately, this tradition of stigma, while present in Alaska, has not become as established as it is Outside.

Bush Alaska is also confronted with the problem of where to hospitalize psychiatric patients. It has been suggested, and even mandated in some areas, that bush hospitals take on a greater role in evaluating and treating those people needing psychiatric hospitalization. However, few bush hospitals are prepared to deliver psychiatric care, and virtually none are prepared to handle a violent psychiatric patient or a psychiatric emergency. The hospitals are small and have limited nursing staffs to handle the relatively small inpatient censes. It is not unusual for bush hospitals to have 2–3 nurses on duty for each of the evening and night shifts—which compose two-thirds of the day. Thus, the number of people needed to handle a psychiatric emergency, such as safely subduing a violent or psychotic patient, would be unavailable. Police or accessory personnel can be summoned, but even if they respond quickly the wait could result in an injury to staff, patients, or both.

Moreover, even the newest bush hospitals usually do not have the facilities needed to contain a violent patient. Few, if any, have "quiet" rooms, and an agitated or suicidal patient is often placed in a regular hospital with a veritable cornucopia of options for self harm at his disposal. Such rooms, equipped with windows, glass, electric and gas outlets, heavy furniture, and sharp objects are by no means safe for patient or staff to contain a difficult patient.

To add to this dilemma, few bush communities have psychiatric staff to aid in the care of their psychiatric patients. Thus, the care becomes the responsibility of the general practitioner and staff, who, while expert in emergency procedures as a whole, may not be prepared or comfortable in dealing with psychiatric issues.

Thus, it would be unreasonable to expect that all bush hospitals care for all dimensions of psychiatric emergencies from their area, even for the first few days. While there are certain facilities, or certain patients who can be adequately treated closer to home, to attempt to treat all would be flirting with danger, and eventual disaster.

So, what would another option be?

Effective care could be provided by centralizing psychiatric hospital care in Anchorage, Juneau and Fairbanks, which would serve as secondary care facilities for the bush. While in general it is useful for patients to be treated close to home, that advantage evaporates if they cannot receive optimum care. The three large cities would be much better able to provide comprehensive psychiatric care. Likewise, this optimal care could only be provided if the psychiatrists caring for the bush patients would keep in close contact by phone with the patient's local physicians. Communication is essential to insure that treatment plans be continued after discharge.

Fortunately, the patient discharged to the bush today has the advantage of having access to community mental health centers which are now well established. The larger bush communities have community mental health centers staffed by masters-level clinicians who provide outpatient services to their town, as well as site visits to surrounding villages. Clinicians travelling to bush villages get to know their patients and families better and can therefore provide better care. Patient's families get to know the caregivers and are more likely to trust the clinician and call on him in an emergency. Consultation is also provided to local hospitals by these clinicians, and emergency psychologic care is offered as well.

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Ongoing psychiatric services are not always available. Psychiatric services could be provided better if more psychiatrists followed the lead of their counterparts in the community mental health clinics. If psychiatrists in the urban centers of Anchorage and Fairbanks would venture to the community mental health center every few months and have weekly phone conferences between visits, they would get to know the patients and their treatment plans, as well as the skills of their colleagues, and could become much more a part of bush psychiatric care. By knowing each other's skills, many matters could be easily handled more quickly and with more expertise. Diagnostic questions could be evaluated through case discussion, and the resultant improved services would decrease the need for many hospital admissions.

In addition to the improvement in care using the community mental health system as the center of a system of care would reduce costs. Paying for transportation and consultation fees for a psychiatrist would be expensive; however, the improved care would abort many admissions, thereby paying for the service. It has been demonstrated repeatedly that community based treatment programs are more effective (1-3), and are cheaper (3,4) than systems that are based on hospital or institutional care. For a community-based treatment system to work, it must have the financial support of the state and the community. It must also have the support of the medical community with the psychiatrist willing to provide services to the bush on a routine basis and willing to be a "phone call away" to lend emergency assistance. Alaska, with its high rates of suicide and alcoholism (5), needs a community-based treatment system to attack these public health problems.

Another great problem facing the delivery of comprehensive psychiatric care to the bush is the confusing array of health care systems. All systems attempt to provide services, but all run on their own course with little interactive planning. Living in the bush in Alaska and being psychiatrically ill or disabled, a person is likely to be eligible for care by the Indian Health Service, a federal agency, but would be hospitalized at the state hospital in Anchorage. Routine care would be provided by the community mental health center with local, private, state, and federal support. Auxiliary services would be provided by native non-profit organizations and city services, as well as local hospitals which may be federally run, state funded, non-profit, or for profit, etc. In addition to this confusion is the military system—a large presence in Alaska with its own hospital and caregivers. Such an assortment of services at face value may present the notion that much is available, but all too often these organizations seemingly operate in a vacuum with little coordination and planning with other agencies and delivering identical services to the same group of people at the same time.

For any system to work, it must be coordinated. Alaska can no longer afford to have many agencies combatting a problem in their own way without interacting in the planning and coordination of delivery services. The community mental health center serving as the hub of the wheel, with psychiatric input and state and federal support serving as spokes to provide help from the outside, offers the best hope for quality, cost-effective care.

In summary, some problems in the delivery of quality psychiatric care to bush Alaska are unavoidable due to the size of the state. Others are caused by the bureaucracies of the many facilities that staff it. It is unreasonable to expect small community hospitals in the bush to serve as specialty hospitals—i.e. psychiatric facilities. While they may be able to handle less complicated psychiatric admissions, most cases should be forwarded to facilities better able to handle inpatient psychiatric care. The bush community mental health center remains the best bet to coordinate services and function as the primary mental health caregiver in the state. These community mental health centers, however, will need financial and psychiatric assistance to take on this expanded role. The extra cost would be paid for by aborted admissions and better coordination of the bureaucracy surrounding care in Alaska. In this way, the mental health needs of bush Alaska could be better served.

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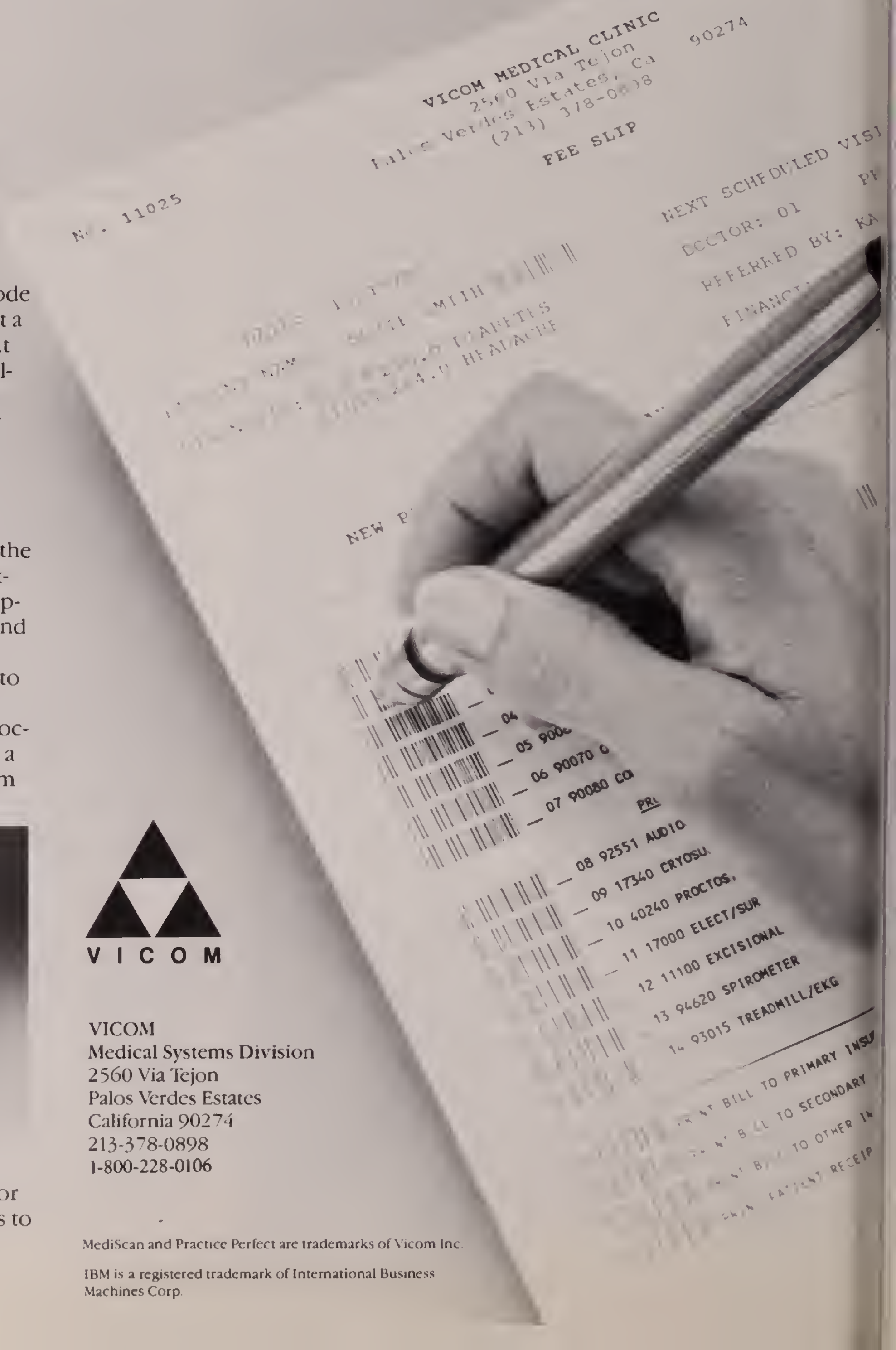

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Special Article

Uncompensated Care Threatening the Future of Alaskan Health Care

by Kaaren Johnson[1]

OVERVIEW

Uncompensated care has become the number one threat to the future availability of health care in Alaska. All health care providers, especially hospitals, physicians, long term care and outpatient facilities, are caring for more and more people who have no way to pay their bills.

A long-range state health policy is desperately needed to protect the disadvantaged while, at the same time, assuring the continued viability of our health care system for all Alaskans. In 1987, the Governor appointed an Interim Commission on Health Care to examine and recommend changes in laws, regulations, and policies necessary to:

- reduce or contain the cost of health care in Alaska; and
- increase access to health care for Alaskans who lack the resources to obtain adequate health care.

The work of the Interim Commission, found in the September 1988 report, *The Best of Care: The Challenge of Providing Health Care to Alaskans,* is an excellent beginning at that difficult task of establishing a State Health Policy.

This article, building on the work of the Interim Commission, describes the issues surrounding uncompensated care and the magnitude of the problem, especially for Alaska. It summarizes solutions being considered in other states or which might be considered for Alaska to manage this problem before it reaches a crisis level where programs are eliminated, facilities close, and patients must seek care "Outside."

Uncompensated care as defined here includes both charity care and bad debt. Many Alaskans are unable to or do not pay for their care. Most are medically uninsured, without health coverage by either private or public sources. They are not necessarily the destitute (who would often be eligible for Medicaid or other public funding); but often are the marginal (those who "fall through the cracks") and include:

- **the uninsurable** — insurance companies will not cover people with certain chronic diseases or conditions regardless of income or financial resources,
- **the working poor and/or uninsured** — those employed in jobs that offer no fringe benefits (or recent labor force entrants whose health policies do not yet apply),
- **the retired** — those who do not have and can not purchase insurance for long term and other chronic care, not otherwise covered by Medicare or major medical insurance.

The tradition of most community providers has been to treat people first and ask about payment second. However, the growth in uncompensated care is contributing significantly to a financial crisis for Alaska's health care services. The costs of uncompensated care keep facilities from funding facility repairs, replacing equipment, or investing in new technology.

If the problem continues, programs and services will be eliminated. Alaskans will be sent outside for their health care. In the worst case, entire facilities may close. Currently, some facilities are reducing staff and experiencing financial losses. While due in part to the state's economic downturn and population losses, this financial drain is reaching crisis levels because of growing levels of uncompensated care.

Uncompensated care stems primarily from cutbacks in government programs, greater unemployment, and the elimination of health insurance benefits by small employers trying to cut costs. Historically, traditional payment sources, (e.g., private insurance and government programs) helped share in the cost of care for indigent patients by including charity and bad debt as a legitimate part of the allowable overhead in their reimbursement of charges (cost–shifting). However, most payors no longer allow that contribution. For example, federal Medicaid rules disallow bad debts and charity to count in determining a facility's reimbursement rate, so such losses must be born solely by the facility!

Nationally, a 1987 congressional report found that 35 million Americans were uninsured. According to the Employee Benefit Research Institute, the "working uninsured" represent 70% of that total, or 24.5 million. This means that one out of six working Americans is not covered for health care expenses by private or public sources. Half of these uninsured workers are concentrated in two industries — retail trade and services. The Batelle Report estimated that in 1985, 44,000 Alaskans were without health insurance.

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[1] 3200 Providence Hospital, Anchorage, Alaska 99508

Left alone with this community burden, the health care provider has a perilous future.

THE ALASKAN EXPERIENCE

Alaskans are facing a serious and growing problem. The recent Report of the Governor's Interim Commission on Health Care concluded that access to health care is one of two areas needing *immediate* attention by policy makers.

Acute Care:

While the problem is not limited to hospitals, information readily available on the magnitude of the problem in Alaska comes from a study of hospitals by the Medicaid Rate Commission (Spring 1988):

- Between 1984 and 1987, the total revenue for Alaskan hospitals grew 30%; yet the amount of uncompensated care increase 100%!
- In 1987, Alaskan hospitals contributed over $20 million or 6.7% of revenue in patient care to individuals who could not pay (See Attachment A).
- By comparison, Washington State hospitals attributed 4.6% of revenue to uncompensated care that year.
- In 1988, the largest hospital in the state, Providence, had $9,800,000, or 8.2% of revenues, in uncompensated care.

Unlike most states, in Alaska there are no public or "county" hospitals specifically funded to provide care for medically indigent, non-Native patients. The burden to care for these patients rests wholely with private community hospitals and clinics. While most rural hospitals have city or municipal ownership, they are not "public" hospitals. As such, they are not designated or funded to care for the poor. The four hospitals in Anchorage, Fairbanks and Palmer are privately owned; yet all provide significant and rapidly growing amounts of care which is uncompensated.

Long-Term Care:

For long-term care facilities in Alaska, uncompensated care is also a growing concern.

Historically, Medicaid has been the primary source of payment for long-term care. Payment programs for the middle income elderly needing long-term care services have been non-existent. Retirement benefits, pensions, and savings are often insufficient to pay for nursing home care. Yet, the higher incomes of the middle income elderly exceed Medicaid limits, leaving these seniors ineligible assistance. Until recently, private insurance for long-term care was not available for purchase. Policies now offer only minimal coverage or disallow major categories of illness. Medicare, the medical care program for the elderly, has never paid for long-term care (and contributes to only about 2% of a facilities's revenues).

Alaska has traditionally assisted with funding for long-term care through Medicaid, General Relief Medical (GRM), Catastrophic Illness (CI) funds, and support for the Pioneer Homes. The state has recently not funded GRM and CI, leaving health facilities with no choice but to absorb the costs of care or deny admission to those without sufficient funds.

The Alaskan Pioneer Home system offers long-term nursing care. As the Interim Commission report discusses, several of its policies are in direct conflict with a prudent state financial policy. The state has assumed total responsibility for the payment of care in the Pioneer Homes rather than take advantage of the federal Medicaid program which offers a 50/50 match of federal and state dollars. The state gives admission priority to the "destitute" pioneer, the same person who would have been eligible for those matched Medicaid funds at a private nursing home. The middle income elderly gets left out, with no room at the Pioneer Home and no funding for other facilities.

Uncompensated Care: One System's Example

The impact and threat of uncompensated care on the financial health of the acute and long-term care facilities is enormous. For example, in 1988, the Sister of Providence owned three programs—Providence Hospital, Our Lady of Compassion Care Center, and Home Health Inc.; and managed the Mary Conrad Center and the Alaska Treatment Center. That same year, the three owned facilities alone had **$11,238,000** of uncompensated care.

	Uncomp. Care (estimated)	**% of Revenue**
Providence Hospital	$10,400,000	8.3
Our Lady of Compassion	820,000	4.7
Home Health, Inc.	18,000	2.0
TOTAL	**$11,238,000**	

PROPOSED NATIONAL AND STATE INITIATIVES

There are a variety of proposals being considered nationally and in local areas throughout the country to respond to the problems of increased uncompensated care and financing of health care for the poor. A 1987 federal report, "Health Care Policy Agenda for the American People," recommends that both public and private sectors must participate in finding the solutions to this problem (See Attachment B). In consideration of the options and proposals, the report recommended that:

> "adjustments and corrections to the current system(s)
> and/or
> local or state options for program designs
> are preferred to
> major new national initiatives or direct subsidization."

Several national initiatives which are currently being considered include:

- **National Health Insurance:** In a recent editorial, the New England Journal of Medicine has recommended that it is time for the United States to enact a program of national health insurance. There are a variety of alternative models which could be considered. This is not a new idea. It is significant, however, that the New England Journal of Medicine has taken up the idea. There has not been much support for national health insurance in the past because of concerns about the cost and financing of most proposals.
- **Mandatory Insurance Coverage:** One way of ensuring uniform coverage of all employed workers is to require all employers to provide health insurance coverage. While this is the most direct way to ensure coverage, it raises several problems. Two thorny issues are whether mandatory health insurance would price some minimum wage workers out of the market, and whether small employers could financially comply with the provisions.
- **Trust Fund for Indigent Care:** Proposed federal legislation would levy a 1% excise tax—paid by employers—on the cost of their health insurance plans. This would raise about $500 million in fiscal 1988 to establish a trust fund for indigent care. Hospitals seeking funds would submit audited financial information to determine their share of uncompensated care.
- **Modifications to Medicaid/Medicare Programs:** A variety of options for changes in federally funded programs and changes in funding levels are periodically proposed. For example, some uninsured workers are substantially below the poverty line, but they do not qualify for Medicaid. One approach is to allow poor workers to buy into Medicaid by paying premiums on a sliding fee scale related to income level.

OPTIONS FOR ALASKA TO CONSIDER:

There are a number of options which Alaska might consider. Some could be readily instituted and would have an immediate effect on the delivery and funding of health care by making adjustments to the current system and/or financing. Several options are discussed in the Interim Commission report, others are being considered by other states.

- **State Insurance Plan:** Provide health insurance for either all Alaskans or to the working poor or elderly. In Alaska there are a large number of small employers who can not afford to offer health benefits to their employees. The Interim Commission report discusses several methods for expanding and financing insurance coverage. Such options might be helpful to small employers as well as the uninsured. For example, the state might extend the current Long Term Care insurance program provided to retired state officials to all residents 50 years and older. Permanent Fund Dividend checks could be accepted as payment for insurance. Examples of the State Insurance or Basic Health Plans are being considered in Washington and California.
- **Insurance Risk Pool:** Create a risk pool to provide health insurance to residents of the state without coverage because of pre-existing conditions. Oregon established but did not fund such a pool.
- **Employer Tax Credits:** Offer tax credits for small employers to provide health benefits to their employees. In Oregon, employers are given up to $25 per month per employee to provide insurance coverage.
- **Generate Additional Funds:** Develop a fund or indigent pool designated to finance care of the medically indigent, through such sources as:
  - Sales tax on commercial services, including advertising (Florida)
  - Assessments on hospitals and municipalities (South Carolina)
  - Add-on surcharges to hospital Medicare payments (New York)
  - Property taxes (Nevada)

  Modify Pioneer Home Benefits: There are several ways in which the Pioneer program can be modified to be much more cost effective and to allow the state to take advantage of significant additional funds (see below).
- **Utilize Medicaid to the Fullest:** Select several options offered by Medicaid, to take advantage of the 50/50 federal match:
  - Adopt the Medically Needy option

- Extend Medicaid coverage to families terminated from AFDC because of their earnings to the maximum extension permitted by federal law, as Oregon has recommended.

RECOMMENDATIONS:

While the state should study the various solutions being considered by other states, some would *not* be feasible or successful in Alaska. For example, the citizens of Alaska are *not* currently interested in increasing taxes as a source of increased revenues. State law does not allow excise taxes to be designated for a specific purpose (for example, a cigarette tax to be designated for exclusive use to fund health care). State insurance plans or risk pools might have merit, although the small population base could make the plan expensive or cumbersome if it is not carefully designed.

Several recommendations of the Governor's Interim Commission on Health Care should be implemented, specifically; (1) expand Medicaid to include the Medically Needy Option, and (2) modify the Pioneer Home Program to optimize the use of state and federal funds for all senior citizens.

The Medically Needy Option Under Medicaid:

Medicaid is a federal program for health care for the poor which includes federal and state funding. Medicaid has a number of mandatory programs, but it also offers some optional programs which each state can select and modify to meet its own priorities and needs. One of the most attractive of these options is the "Medically Need Option," because the Medically Needy Option:

- is an excellent way to serve the poor in need of care who are without resources but not otherwise eligible under Medicaid;
- maximizes the use of federal matching dollars;
- provides that the state can tailor the coverage to meet its own priorities and budget limits, and
- allows the client to spend down their income on their medical expenses in order to become eligible for Medicaid.

Modification of the Pioneer Home Program:

There are several ways in which the Pioneer Home Program can be modified to reduce its total dependency on state funds. While these changes are controversial for the pioneers of Alaska, they may be the only way to assure the long-term continuity and fiscal viability of the program. Such changes include:

- consider economic participation on the part of the residents on a sliding scale according to the assets and income of the residents;
- modify the program so the nursing home beds are Medicaid eligible to best utilize the federal matching funds. This may mean a change in residency requirements;
- establish admission criteria which would encourage the use of Medicaid funds and placement in Medicaid facilities by those pioneers who are eligible for Medicaid.

CONCLUSION

A long-range, state-wide health policy is necessary which will

- promote and protect the public health,
  - assure equitable access to high quality health care for all Alaskans,
    - encourage a shared responsibility for health care,
      and
      - assure the continuity, growth, and economic viability of the health care institutions, physicians, other providers and the various other health care programs.

Alaska is blessed with excellent health care services. We all must work together to assure that health care continues to be available and affordable to all Alaska's citizens.

ATTACHMENT A
MEDICAID RATE COMMISSION
STATS: Percent Uncompensated Loss & Profit Margin by Facility Fiscal Year

FACILITY	TOTAL PATIENT SERVICE REVENUE	BAD DEBTS CHARITY	% UNCOMP LOSS	NET INCOME	N.I./T.R.
Bartlett Memorial Hospital:					
1986	$12,682,939	$210,999	1.66%	$204,024	1.61%
1987	12,982,923	924,141	7.12%	(351,113)	-2.70%
Central Peninsula Hospital:					
1986	$10,185,840	$674,433	6.62%	$1,463,790	14.37%
1987	10,625,224	954,964	8.99%	2,098,899	19.75%
Charter North:					
1986	$11,442,039	$645,377	5.64%	$1,408,603	12.31%
1987	11,374,218	583,333	5.13%	1,519,347	13.36%
Cordova Community Hospital:					
1986	$1,225,224	$35,804	2.92%	$65,340	5.33%
1987	1,659,092	29,480	1.78%	(342,295)	-20.63%
Fairbanks Memorial Hospital:					
1986	$35,264,058	$1,496,134	4.24%	$ 719,513	2.04%
1987	36,881,080	2,729,264	7.40%	1,190,241	3.23%
Faith Hospital:					
1986	$657,735	$65,471	9.95%	$65,383	9.94%
1987	N/A	N/A	N/A	N/A	N/A
Humana Hospital:					
1986	$58,897,501	$3,449,440	5.86%	$2,825,979	4.80%
1987	63,500,602	3,044,980	4.80%	3,600,774	5.67%
Ketchikan General Hospital:					
1986	$9,388,997	$312,152	3.32%	$387,348	4.13%
1987	9,512,408	331,634	3.49%	362,651	3.81%
Kodiak Hospital:					
1986	$5,579,432	$266,593	4.78%	$ 274,259	4.92%
1987	6,484,413	301,491	4.65%	1,063,486	16.40%
Norton Sound Regional Hospital:					
1986	$4,583,385	$154,558	3.37%	$278,368	6.07%
1987	5,250,928	312,909	5.96%	518,683	9.88%

FACILITY	TOTAL PATIENT SERVICE REVENUE	BAD DEBTS CHARITY	% UNCOMP LOSS	NET INCOME	N.I./T.R.
Petersburg General Hospital:					
1986	$1,859,070	$34,401	1.85%	($231,125	-12.43%
1987	1,936,256	8,859	0.46%	(153,576)	-7.93%
Providence Hospital:					
1986	$103,223,000	$6,457,000	6.26%	$1,101,000	1.07%
1987	117,773,000	9,617,000	8.17%	1,489,000	1.26%
Seward Hospital:					
1986	$1,515,061	$ 57,178	3.77%	$149,443	9.86%
1987	1,643,853	129,524	7.88%	(64,049)	-3.90%
Sitka Community Hospital:					
1986	$2,711,874	$ 71,650	2.64%	($247,180)	-9.11%
1987	4,047,720	134,116	3.31%	399,162	9.86%
South Peninsula Hospital:					
1986	$5,084,850	$427,045	8.40%	($646,804)	-12.72%
1987	5,064,974	183,323	3.62%	($194,114)	-3.83%
Valdez Community Hospital:					
1986	$612,140	$59,839	9.78%	($21,250)	-3.47%
1987	807,342	83,382	10.33%	32,482	4.02%
Valley Hospital:					
1986	$12,107,000	$970,000	8.01%	($297,000)	-2.45%
1987	$12,210,000	957,000	7.84%	1,086,000	8.89%
Wrangell General Hospital:					
1986	$1,657,531	$27,607	1.67%	$229,690	13.86%
1987	$1,788,634	4,347	0.24%	($138,353)	-7.74%
Total All Facilities:					
1986	$278,677,676	$15,415,681	5.56%	$7,662,429	2.75%
1987	303,542,667	20,329,747	6.70%	12,117,225	3.99%

ATTACHMENT B
RECOMMENDATION FROM "HEALTH CARE POLICY AGENDA FOR THE AMERICAN PEOPLE"

According to a 1987 federal report, "Health Care Policy for the American People," the problem of access to health care for all Americans is extremely complex and a wide variety of activities must be initiated if an overall solution is to be created. Both the public and private sectors must be encouraged to participate in the donation of resources to improve access to services.

The report recommended several broad categories of action be considered in addressing the issues nationally. These include:

- Where appropriate, incentives should be developed to stimulate and support increased involvement by the private sector to give care to those who otherwise would not have access to such care. These incentives might be provided under existing or modified tax laws. The incentives would apply to various providers, physicians, dentists, hospitals, long–term facilities and home health agencies, etc..
- Individual health care providers, institutions and philanthropic organizations should be publicly and privately commended, rewarded, and encouraged to maintain and expand, where appropriate, their contributions to public health services programs.
- There should be sustained, enhanced public reimbursement of facilities and professionals that offer and provide voluntary care.
- The Medicaid program should be made more equitable from state to state. States should be required or encouraged to include not only their "categorically" eligible residents, but also their medically "needy" residents. (Currently, Alaska is one of the 16 states which does *not* provide coverage through Medicaid for the *medically needy*).
- Existing shortcomings must be corrected in the public (e.g. Medicaid and Medicare) systems which create gaps or prohibit access to care.
- Private sector groups should work with the public sector to address the issues of health care coverage for the unemployed who have lost health benefits and for individuals who currently "fall through the cracks" of the health care system.

ALASKA STATE MEDICAL ASSOCIATION
and
FEDERAL AVIATION ADMINISTRATION
sponsor the
44th ANNUAL CONVENTION
JUNE 6 - 11, 1989

A Whole Physician in a Healing Universe

Registration/Information

Alaska State Medical Association
Jeanette McCart, Executive Assistant
2401 E. 42nd Avenue, Suite 104
Anchorage, Alaska 99508
Phone (907) 562-2662

ASMA/FAA cosponsored meeting.

Registration Fee: $250.00 for Non-member physicians. This fee entitles physicians to attend all CME sessions. No fee is required for ASMA members or FAA registrants.

Please register prior to May 5, 1989.

Location & Date of Meeting

Sheldon Jackson College
Sitka, Alaska
June 6-11, 1989

Continuing Medical Education

Over 25 hours of scientific medical education will be provided by nationally recognized physicians.

Accommodations

Over 300 rooms have been reserved at special rates in Sitka, all within walking distance to Sheldon Jackson College and the Centennial Building.

Activities

Excitement will be high in Sitka! Attendees can enjoy the Sitka Music Festival, New Archangel Dancers, relax while cruising to an island for a salmon/halibut bake, tour historic Russian America, meander through Totem National Park, and for the adventureous, run for fun with Alaska's fastest (and not so fast). The grand finale to five fun-packed days will be the President's Dinner Ball.

Family Fun

A full slate of spouse and children's activities are being planned by the ASMA Auxiliary. Babysitting will be available.

Transportation

Alaska Airlines provides daily jet service from Seattle, Anchorage, and other Alaskan cities. Phone 800-426-0333 for reservations. For local info, check phone book.

Ferry service is available from Seattle and S.E. Alaska. Phone (800) 642-0066. Shuttle service and rental cars are available in Sitka.

Deposit/Cancellation

Non-member physicians must send $50 preregistration fee by May 5. Full refunds will be made upon request prior to May 12, 1989. No refund thereafter.

Schedule of Events

Tuesday - June 6, 1989

8:30-4:00 p.m. - AME Workshop
1:30 p.m.- ASMA Council Meeting
3:00-5:00 p.m.- House of Delegates
5:00 p.m.- Dinner-on-your-own

Wednesday - June 7, 1989

8:00-9:00 a.m. - Breakfast
Keynote speakers
9:00-12:00 noon-Scientific Session
1:30-5:00 p.m.-Scientific Session
5:15-6:15 p.m.-Exhibitors' Reception
6:15 p.m.-Dinner-on-your-own
8:15 p.m.-Sitka Music Festival
(private performance)
Survival Training throughout Day

Thursday - June 8, 1989

8:00-12:00 noon-Scientific Session
12:15-1:15 p.m.-Lunch with Speaker
1:30-5:00 p.m.-Scientific Session
6:30 p.m.-Russian Dinner
w/Entertainment

Survival Training Throughout Day

Friday - June 9, 1989

8:00-11:45 a.m.- Scientific Session
12:00 noon - Lunch w/ Speaker
1:30- 4:45 p.m.- Scientific Session
5:15 p.m.-Cruise to Salmon/Halibut
Bake on Island

Saturday - June 10, 1989

6:30 a.m.- AAFP Fun Run
8:00 a.m -Breakfast - Charles Chappell
Associate Director for
Science, NASA
8:50-9:45 a.m - Scientific Session
10:15-11:45a.m.-Panel Discussion
12:00 noon - Lunch w/ Speaker
1:30-5:00 p.m. - House of Delegates
6:30 p.m. - President's Banquet&Ball
Keynote Speaker - Chuck Berry,
Medical Director of NASA
Entertainment & Awards

Sunday - June 11, 1989

9:00 a.m.- Council Meeting

REGISTRATION FORM

NAME: ______________________ DATE: __________
ADDRESS: ______________________________
______________________ ZIP: __________
NUMBER OF ADULTS IN PARTY: __________
SPOUSE'S NAME: __________
NAMES & AGES OF CHILDREN: __________ AGE: __________
(in attendance)
__________ __________
__________ __________

HOW AND WHEN ARRIVING: ______________________
Send check or money order with Registration Form to: Alaska State Medical Association
2401 E. 42nd Avenue, Suite 104 Anchorage, AK 99508

News from the American Society for Circumpolar Health

* * * * * * * * * * *

The board has been organizing and planning for several major health conferences.

- September 1989 – Annual Meeting of ASCH during AAAS meeting – Fairbanks, Alaska
- September 1989 – Annual Arctic Division, American Association for Advancement of Science (AAAS) – Fairbanks, Alaska
  We will have a major session on arctic health science.
- May 1990 – 8th International Congress on Circumpolar Health – Whitehorse, Yukon, Canada
- May 1990 – General Assembly Meeting – International Union for Circumpolar Health (during ICCH, Whitehorse, Yukon, Canada)
- September 1990 – Annual Meeting of ASCH
- Major health sessions during AAAS, Anchorage, Alaska

MARK YOUR CALENDARS! PLAN TO ATTEND!

* * * * * * * * * * *

The Alaska Science Foundation has published its first annual report (January 1989) and has put out a request for proposals. Although the April 1 deadline for letters of intent will have passed prior to publication of this issue, we will watch with great interest what health research proposals are received and funded.

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American Society for Circumpolar Health Officers

October 1, 1988 – September 30, 1989

President	John Middaugh, M.D.
Vice President/ Scientific Director	Robert Fortuine, M.D.
Secretary	Frank P. Pauls, Ph.D.
Treasurer	Helen D. Beirne, Ph.D.
Board of Directors	Anne Lanier, M.D.
	William J. Mills, M.D.
	Philip O. Nice, M.D.
	Brian J. McMahon, M.D.
Honorary Members	Wayne W. Myers, M.D.
	C. Earl Albrecht, M.D.
	Frederick A. Milan, Ph.D.

ASCH now has 152 active members, 103 having paid their 1989 dues. We recently have welcomed several new members from the U.S. Navy Antarctic Research Program.

* * * * * * * * * * *

ASCH is now distributing to all ASCH members *Arctic Medical Research,* the official journal of the International Union for Circumpolar Health, a publication of the Nordic Council for Arctic Medical Research.

* * * * * * * * * * *

The International Union for Circumpolar Health has sponsored a meeting on "Tobacco and Health" in Yellowknife, Northwest Territories, Canada on April 4–6, 1989. During this meeting there also will be held a meeting of the International Union for Circumpolar Health. Dr. Robert Fortuine, M.D., one of the two ASCH delegates will attend the meeting, representing the ASCH.

* * * * * * * * * * *

The International Union for Circumpolar Health will have many important items to discuss in Yellowknife. Among them are the relationship of the IUCH to the newly forming International Arctic Science Committee (IASC), the relationship of the IUCH to the International Council of Scientific Unions (ICSU), and the relationship of the IUCH to other new organizations.

Several organizations have expressed an interest in establishing formal ties with the IUCH including the Society for Medical Research in Greenland, Icelandic Society for Circumpolar Health, and the German Society for Polar Research.

* * * * * * * * * * *

The Conference of Arctic and Nordic Countries on Coordination of Research in the Arctic took place in Leningrad, USSR in December 1988. More than 500 participants attended representing all disciplines involved in arctic science and research. The following statement was agreed to by the participants at the meeting but not released until approved by the appropriate governments.

Statement on Circumpolar Health Studies

From the medical participants in the International Conference of Arctic and Nordic Countries on Coordination of Research in the Arctic, Leningrad, U.S.S.R., December 12–15, 1988.

We recognize that human health has the highest priority, and that the population groups of the North, the indigenous peoples and the migrants, have some unique health problems of growing concern.

Both groups are confronted with a new culture. The acculturation problems are much more difficult for the indigenous population constituting probably one of the main causes of the health problems, e.g., high rates of behavioral disorders like alcoholism, suicide, violence, and venereal disorders.

The indigenous population groups still suffer from some traditional diseases (contagious and parasitic diseases) but are also confronted with serious health problems related to modernization and industrialization (occupational injuries and diseases) and other diseases such as some types of cancer and cardiovascular diseases. Thus, some groups of the population have 20–30 times the incidence of suicide, alcohol addiction, nasopharyngeal carcinoma, iron deficiency anemia, and pneumococcal meningitis as compared to other populations.

The migrants have biological, psychological, and cultural adaptation problems caused by the climate, darkness, and other factors.

Circumpolar health and research is relevant to all of us and may give clues to the cause of some of the most serious diseases of the industrialized world (e.g., atherosclerosis, cancer, etc.).

Considering that biomedical research in circumpolar health is inadequate and is lacking in some areas of high priority, the participants of this conference recommend:

1. The creation of an Advisory Committee for Circumpolar Health Studies to the planned International Arctic Science Committee. This advisory committee can be based on the principles of the existing International Union for Circumpolar Health;
2. Increased biomedical research in areas of high priority;
3. Bilateral and multilateral international cooperation in all areas of mutual benefit;
4. The establishment of international working groups in the following high priority areas:
    a. addictive disorders;
    b. ecology and disease
    c. cancer and infectious disease;
    d. mental health;
    e. maternal and child care; and
    f. pollution and environmental hazards.

    International working groups on circumpolar cancer and hepatitis B are already functioning within the International Union for Circumpolar Health;
5. Urgently needed funding for the immediate development of integrated basic and clinical research in the circumpolar regions.

YOCON®
YOHIMBINE HCl

Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:
1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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ALASKA
PUBLIC HEALTH ASSOCIATION

News from the Alaska Public Health Association

President's Message

A recent Anchorage Times headline read "Quake Commemoration will be held in March." It struck me how many ways ALPHA and its members are shaping the public health of Alaskans. No, I'm not about to claim that we have stopped killer earthquakes in Alaska. But we did go on record at last year's ALPHA Health Congress, resolving to designate March earthquake observance month in order to increase public awareness and preparedness. Now Governor Steve Cowper has declared march as "Earthquake Awareness Month."

There are other major areas where we are also making a difference. Injury control is one. Board members Carl Hild, Tom Nighswander, MD, and I met with Senator Ted Stevens in late December, seeking his support for injury control issues. We were well received and the Senator is committed to working to reduce injuries. I received a phone call from Kathryn McCarter, Legislative Affairs Director of the American Public Health Association, thanking us for our efforts in injury control and thanking Senator Stevens for his willingness to champion increased injury control appropriations if the Senate this year! We will continue working with the Senator and his staff to establish an injury control clearing house in Alaska.

We are making an impact on the state level, as well as the national level. Because we are the recognized organization of people concerned with public health in Alaska, the Board was approached to provide input in developing the request for proposals to the Alaska Science and Technology Foundation. The Foundation was established in 1988 to fund applied research for Alaskan economic development and public health. Six million dollars may be available. The board recommended proposals be solicited in the areas of 1) injury control, 2) health promotion/disease prevention especially aimed at children, and 3) establishment of a unified health policy. The Foundation is including our first two recommended areas in their April 1 deadline for proposals.

We continue to advocate and agitate for a state health policy. The respondents to our mini-survey, which was sent to 20 people in the fields of injury control, mental health, and alcohol reported unanimous approval for the concept of a state health policy.

In my last Message, I reported that Myra Munson, State Commissioner of Health and Social Services, was requesting that her division heads discuss this and formulate a Department strategy. Commissioner Munson has stated, "almost everyday someone asks me about the status of health planning in the Department. I consider that a victory in and of itself. It means that advocates have not lost confidence about my interest and that there is still energy and commitment to make sure it really happens; and that everyone knows it takes persistence to make planning and goal setting stay on an already crowded agenda. There is progress, although it is slower than I wish."

"Preliminary work is underway to design a process for development of DHHS long term goals. The outcome of this necessary work will be a reduced version of a much needed major State Health Policy. In conjunction with this effort the Division of Public Health hopes to take a leadership role in guiding the DHSS Goals project toward a state health policy. We hope the workplan for these activities will be completed by the end of the summer." We have invited Commissioner Munson again to address the ALPHA Health Congress on April 24 and discuss health policy development with us.

While the unified health policy is being formulated, the legislature is moving on significant public health issues. The ALPHA Health Issues Committee, under Tom Nighswander's direction, has kept the membership informed and motivated to communicate with the legislature. As of this writing, seat belt legislation had passed in the House and was still moving.

ALPHA may be the only APHA affiliate with significant international relationships. We again had the opportunity to host Soviet visitors and learn more about our neighbors to the west. On February 23, we had 47 members and guests at the Anchorage Brown Bag meeting to hear Dr. Putilov and Dr. Krivoshekov of the Soviet Academy of Medical Sciences, Siberian Branch, discuss "Circadian Rhythms, Biorhythms and Seasonal Affective Disorder." Through better understanding of each other and our common health concerns, we're making an impact on health internationally.

Flyers for the ALPHA Health Congress have arrived.

The Congress **will be held** April 24-26 **at the** Egan **Convention Center,** Anchorage. Once again the Congress is a coming together of members of the major health-related organizations to exchange information and concerns. Under Susan Jones' leadership, the committees have planned an excellent program featuring nationally known authorities, Eula Bingham, PhD, contributor to The Future of Public Health 1988, and Marian Arlin, RD, MS, author of The Science of Nutrition, former Alaskan, and former ALPHA board member! Many members will also present papers on major programs throughout the state. Resolutions will be presented to set the course and philosophy of ALPHA. It will be the major public health meeting of the year.

We are an effective voice for the health of Alaskans. We have built on past efforts and will continue to build in the future. May our influence continue to be a positive force. We are more effective together than any of us can be separately.

Betsy Nobmann
President
Alaska Public Health Association

Opinion

Are the Fish in Ship Creek Safe to Eat?

By Jennifer H. Christian, M.D.

Recent public discussion in Anchorage over the removal of warning signs on Ship Creek failed to highlight a difference between public health and environmental protection decision-making.

The signs were put up in 1985, when considerable uncertainty existed over the amount of pollution in Ship Creek. The extent of PCB contamination of the Standard Steel yard was still being uncovered. Preliminary studies of the creekbed indicated contamination by PCBs which can enter the food chain.

This was worrisome news that warranted a response from the Municipality. Was this our first warning of a major contamination? Maybe the pollutant load was so high as to represent an acute hazard. The decision to post the warning signs was prudent. The signs read: "NOTICE; The Municipality of Anchorage recommends against the eating of fish from these waters because of chemical contamination of stream sediments."

In 1989, four years later, we were asked whether the evidence at hand required us to continue to caution the public against eating fish caught in Ship Creek. Our decision to take down the signs does not mean we think the creek is sparkling clean; it means we think the fish are safe to eat.

Since 1984, several studies have been done on Ship Creek, studying adult and just-hatched fish, and little wiggly things that live in the streambed (called macroinvertebrates). All the results are now in and have recently been reviewed by the Municipality. These studies have confirmed that there is pollution in the creek.

But the studies have also been reassuring, because the pollution is not massive. Although the pollutant load in the creek may be high enough to kill some of the fish hatchlings, there are still baby fish there. The Department of Fish and Game still uses Ship Creek as a hatchery stream. Furthermore, even the most sensitive macroinvertebrate species are still in the creek, instead of only the few hardiest kinds.

This finding reminds us that "polluted" is a relative term; while the levels of the PCBs in the baby fish are high enough to kill some of them off (which is bad), the macroinvertebrates are doing well (which is good).

No doubt, the scientific community will wish to continue studying this creek, and many other creeks, in order to understand more and more precisely the nature of pollution and its interaction with the creek ecosystem. The studies already done are part of a nationwide survey of bio-accumulation, the gradual increase in pollutants that may threaten the long-term health of our planet.

However, the fact that Ship Creek is polluted does not necessarily mean that eating fish caught in the creek is a public health hazard. Our review team addressed this as a separate issue. We used analytical techniques from biology, microbiology, engineering, toxicology, biostatistics, epidemiology and medicine in the review.

Public discussion of our review process neglected to mention our consultation with the U.S. Food and Drug Administration (FDA). We talked to the FDA's expert on dioxins, as well as with their Washington, D.C., program office. In recent years, the discovery of dioxins in fish from the Great Lakes commercial fisheries spurred a review by the FDA of the safety of dioxins in food. The dioxin levels found in mature Ship Creek fish are below the level the FDA accepts in Great Lakes fish.

To be more specific, let's discuss the levels, without getting too technical. The FDA suggests that fish containing 25 to 50 parts per trillion (ppt) be eaten only once or twice a month. Ground up whole fish from Ship Creek contained less than 5 ppt. We have recently learned that the State of Michigan requires advisories to the public for dioxin levels of about 10 ppt, and

Ontario, Canada, uses 20 ppt.

Pregnant women are a special concern because of the fragile developing fetus. The State of Michigan recommends that pregnant women eat no more than one fish meal a month containing more than 1.0 ppt. Levels of dioxin in Ship Creek are about 4 to 5 ppt. However, whole fish levels of dioxins are about eight times levels found in edible portions. That would mean levels of 0.8 ppt in fillets of Ship Creek fish. And cooking usually removes 40 to 70 percent of the dioxin.

Another simple but important point: Most fish caught in Ship Creek are salmon which have spent their adult lives in open ocean.

What if someone becomes ill from eating a fish out of Ship Creek? That person will not be ill because of PCBs or dioxins from that one or even an occasional fish. The risk to health from dioxins even at levels above those recommended by the FDA are largely theoretical. These risks presume steady doses of dioxins over a period of years before they would have any effect. In addition, the human data we have to date show no adverse effect on reproduction or cancer. These data come from industrial workers exposed to dioxins and citizens accidentally overexposed through industrial accidents or pollution.

Future information cannot be the basis for today's decisions. The fish are safe to eat, based on the best knowledge available to us at this time. Caution is appropriate, but crying wolf is not. I believe it is safe to eat the fish, and will eat them myself if one of you will (legally) catch one for me.

Dr. Christian is Medical Officer, Department of Health and Human Services, Municipality of Anchorage. She also is a private consultant in occupational medicine in Anchorage.

Comprehensive Health Survey Project Underway!

The problems adolescents are facing are often difficult, even deadly. They include accidents due to drunk driving, increasingly common drug abuse, unplanned pregnancies, physical and sexual abuse, and a growing suicide rate. These complex problems are gaining national attention, but how serious are they in Alaska?

No one knows for sure, but finding out is one goal of the Adolescent Health Survey Project, now being conducted throughout Alaska.

The purpose of this project is to develop a statewide database on the health status and concerns of Alaska's youth. With a reliable database, programs can be developed to help our teens.

The impetus for this unique project comes from the Governor's Interim Commission on Children and Youth and other sources. It is a cooperative effort of the Alaska Department of Health and Social Services, Division of Public Health, and the Alaska Area Native Health Service. The project is funded by the Alaska Legislature and the Indian Health Service.

In order to meet the challenge of promoting the health and well-being of Alaska's youth, comprehensive information FROM THE ADOLESCENTS THEMSELVES must be available to educators, health professionals, parents, church leaders and others. The existing database on this population is limited in scope. Little or no comprehensive survey data exist.

While there is a body of literature developing on some health and social service needs of our youth, previous surveys often focused on one dimension of the young person's life or were limited in the ability to generalize for district-specific or statewide planning. The current database is also insufficient for the purposes of statewide program planning, health services evaluation, health care research, health and other curriculum development and evaluation, etc.. The process of planning is hampered by fragmented, non-comparable, incomplete or non-existent data.

The Adolescent Health Survey is a comprehensive survey covering the major health issues facing young people today, such as eating behaviors, nutrition, exercise, alcohol and drug usage, mental health, sexual identity and activity, health care utilization, community and family relations, and personal worries and concerns.

The survey is based on a questionnaire which was developed at the University of Minnesota for use with Native youth nationwide and was modified for Alaska. The original survey was used during the 1986-87 school year in over 80 school districts throughout Minnesota. In total, over 35,000 youths in grades 7 through 12 participated in this effort.

Currently, over 80 percent of Alaska school districts have committed to participating in the project and about 15 districts have completed the surveys.

The confidential survey will help school districts, parent groups, planners and others to understand student's thoughts and concerns about their health so that programs can be developed to better serve teens across the state.

Those directly involved with youth know that risk-taking behaviors do not occur in isolation. Furthermore, the need for information from teens is becoming ever more apparent if communities are going to effectively respond to the needs of their young people.

* * * * *

To implement the survey, the Division of Public Health has contracted with Bill Wood as Statewide Survey Coordinator, and Rocky Plotnick-Weller as the Southeast Survey Coordinator. If you have any questions, Bill can be reached in Anchorage at 279-1940 and Rocky in Juneau at 364-3239.

An Active Stance on Passive Smoking

What environmental pollutant affects the largest number of American children? Cigarette smoke! The best estimates are that between 8.7 and 12.4 million American children less than 5 years of age are exposed to cigarette smoke in their homes. These children are passive smokers; when around smokers, they inhale smoke emitted by burning cigarettes. For many years, we have been aware that children of smokers are more likely to become smokers as adults; if both parents smoke, a child is twice as likely to become a smoker. However, recently incontrovertible evidence of the harmful effects of passive smoking has come to light. Important publications on the health effects of passive smoking were released two years ago by both the National Academy of Sciences and the Surgeon General. A brief overview of the major findings in these two documents follows. The two most important health effects of passive smoking in children are lower respiratory illness and chronic middle ear effusion.

Lower Respiratory Illness

There is strong evidence, from 15 years worth of epidemiologic studies, that children exposed to environmental tobacco smoke have increased respiratory illness rates, especially in the first year of life. In general, children whose mothers smoke are twice as likely to have pneumonia or bronchiolitis in the first year than are children whose mothers do not smoke. There appears to be a dose-response relationship between the amount of maternal smoking and the likelihood of the child's developing respiratory symptoms or a respiratory illness.

Chronic Middle Ear Effusion

Exposure to cigarette smoke is associated with increased rates of middle ear effusion in children. This information comes primarily from a case-control study which was done in Seattle and published in 1983. Children with persistent middle ear disease who had tympanotomy tubes placed, were more than four times as likely to live in a household where more than three packs per day of cigarettes were smoked.

Adult Diseases - Lung Cancer and Cardiovascular Disease

The results of nearly a dozen different studies indicate that passive exposure in adult life to smoking by a spouse increases lung cancer risk by about 33 percent. Furthermore, nonsmokers who live with smokers are at a higher risk of death from arteriosclerotic heart disease than those who live with nonsmokers. No information is yet available about how the risk of death from lung cancer or cardiovascular disease during adulthood may be affected by passive smoking during childhood.

Implications for Alaska

Children living in Alaska may have even greater exposures to environmental tobacco smoke than their counterparts in other regions of the country since they may spend more hours indoors during the long winter. The overwhelming evidence linking lower respiratory disease and middle ear effusions to passive smoking in children should inspire us to protect the health of Alaskan children by preventing this hazardous exposure.

Ruth A. Etzel, MD, PhD

Dr. Etzel is a pediatrician/epidemiologist with the U.S. Public Health Service. She has a special interest in the childhood health effects of environmental exposures. Dr. Etzel is currently assigned to the Section of Epidemiology in the Division of Public Health, Alaska Department of Health and Social Services.

Calendar of Events

ALPHA Brown Bag Lunches

1231 Gambell Street
Anchorage, Third Floor
Conference Room, 12:00-1:00 p.m.

May 25, 1989
Teenage Runaways in Anchorage-Alaska
Take a Positive Step Forward
Jane Barnes, Program Coordinator,
Covenant House Project, Anchorage

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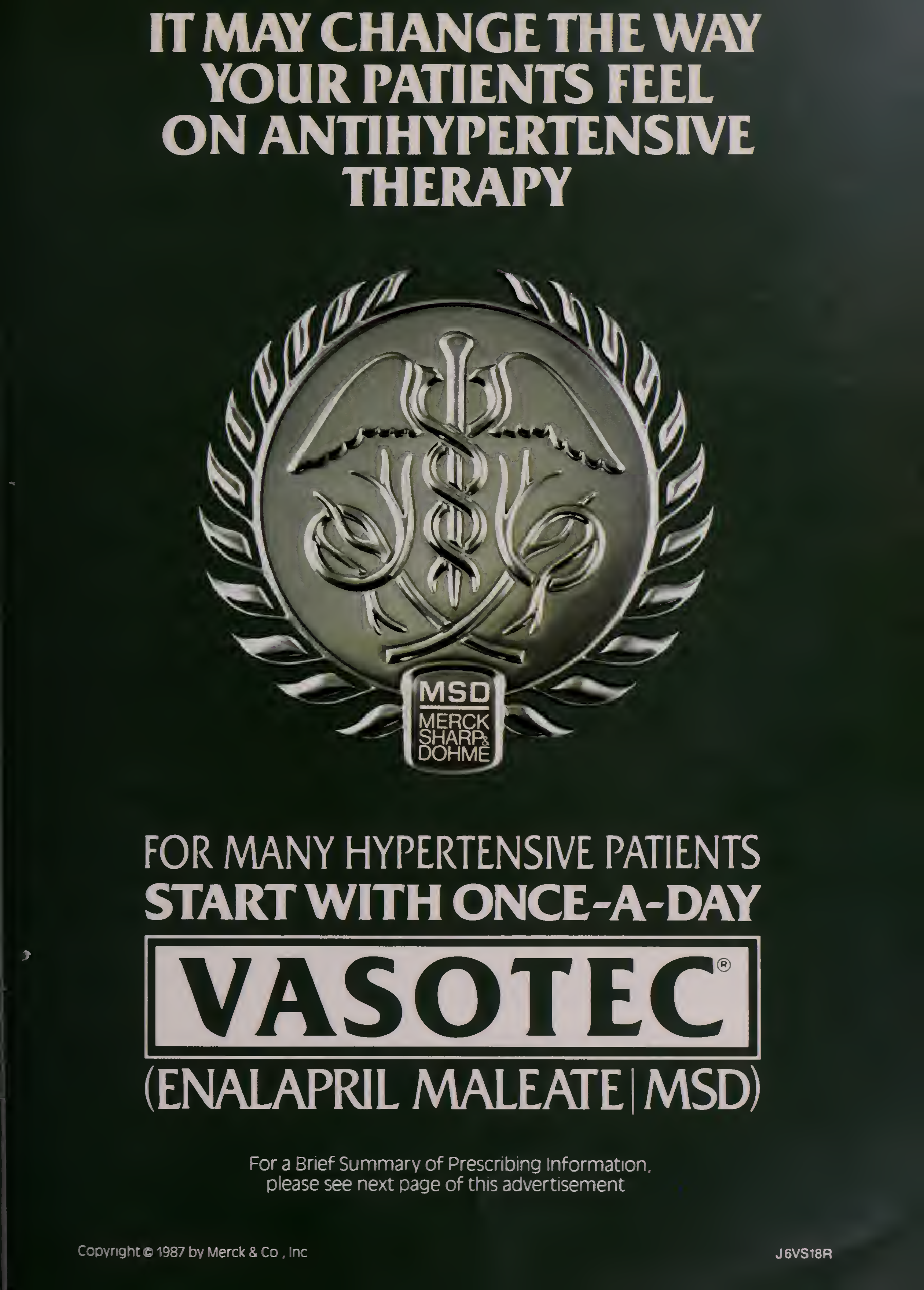

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Contraindications: VASOTEC® (Enalapril Maleate, MSD) is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor.

Warnings: *Angioedema:* Angioedema of the face, extremities, lips, tongue, glottis, and/or larynx has been reported in patients treated with ACE inhibitors, including VASOTEC. In such cases, VASOTEC should be promptly discontinued and the patient carefully observed until the swelling disappears. In instances where swelling has been confined to the face and lips, the condition has generally resolved without treatment, although antihistamines have been useful in relieving symptoms. Angioedema associated with laryngeal edema may be fatal. **Where there is involvement of the tongue, glottis, or larynx likely to cause airway obstruction, appropriate therapy, e.g., subcutaneous epinephrine solution 1:1000 (0.3 mL to 0.5 mL), should be promptly administered.** (See ADVERSE REACTIONS.)

Hypotension. Excessive hypotension is rare in uncomplicated hypertensive patients treated with VASOTEC alone. Heart failure patients given VASOTEC commonly have some reduction in blood pressure, especially with the first dose, but discontinuation of therapy for continuing symptomatic hypotension usually is not necessary when dosing instructions are followed; caution should be observed when initiating therapy. (See DOSAGE AND ADMINISTRATION.) Patients at risk for excessive hypotension, sometimes associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death, include those with the following conditions or characteristics: heart failure, hyponatremia, high-dose diuretic therapy, recent intensive diuresis or increase in diuretic dose, renal dialysis, or severe volume and/or salt depletion of any etiology. It may be advisable to eliminate the diuretic (except in heart failure patients), reduce the diuretic dose, or increase salt intake cautiously before initiating therapy with VASOTEC in patients at risk for excessive hypotension who are able to tolerate such adjustments. (See PRECAUTIONS, *Drug Interactions* and ADVERSE REACTIONS.) In patients at risk for excessive hypotension, therapy should be started under very close medical supervision and such patients should be followed closely for the first two weeks of treatment and whenever the dose of enalapril and/or diuretic is increased. Similar considerations may apply to patients with ischemic heart disease or cardiovascular disease in whom an excessive fall in blood pressure could result in a myocardial infarction or cerebrovascular accident.

If excessive hypotension occurs, the patient should be placed in supine position and, if necessary, receive an intravenous infusion of normal saline. A transient hypotensive response is not a contraindication to further doses of VASOTEC, which usually can be given without difficulty once the blood pressure has stabilized. If symptomatic hypotension develops, a dose reduction or discontinuation of VASOTEC or concomitant diuretic may be necessary

Neutropenia/Agranulocytosis: Another ACE inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression, rarely in uncomplicated patients but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease. Available data from clinical trials of enalapril are insufficient to show that enalapril does not cause agranulocytosis at similar rates. Foreign marketing experience has revealed several cases of neutropenia or agranulocytosis in which a causal relationship to enalapril cannot be excluded. Periodic monitoring of white blood cell counts in patients with collagen vascular disease and renal disease should be considered.

Precautions: *General: Impaired Renal Function:* As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including VASOTEC, may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death.

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine were observed in 20% of patients. These increases were almost always reversible upon discontinuation of enalapril and/or diuretic therapy. In such patients, renal function should be monitored during the first few weeks of therapy

Some patients with hypertension or heart failure with no apparent preexisting renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when VASOTEC has been given concomitantly with a diuretic. This is more likely to occur in patients with preexisting renal impairment. Dosage reduction and/or discontinuation of the diuretic and/or VASOTEC may be required.

Evaluation of patients with hypertension or heart failure should always include assessment of renal function. (See DOSAGE AND ADMINISTRATION.)

Hyperkalemia. Elevated serum potassium (> 5.7 mEq/L) was observed in approximately 1% of hypertensive patients in clinical trials. In most cases these were isolated values which resolved despite continued therapy. Hyperkalemia was a cause of discontinuation of therapy in 0.28% of hypertensive patients. In clinical trials in heart failure, hyperkalemia was observed in 3.8% of patients, but was not a cause for discontinuation.

Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with VASOTEC. (See *Drug Interactions.*)

Surgery/Anesthesia. In patients undergoing major surgery or during anesthesia with agents that produce hypotension, enalapril may block angiotensin II formation secondary to compensatory renin release. If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion

Information for Patients:

Angioedema. Angioedema, including laryngeal edema, may occur especially following the first dose of enalapril. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to take no more drug until they have consulted with the prescribing physician.

Hypotension: Patients should be cautioned to report lightheadedness especially during the first few days of therapy. If actual syncope occurs, the patients should be told to discontinue the drug until they have consulted with the prescribing physician

All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduction in fluid volume. Other causes of volume depletion such as vomiting or diarrhea may also lead to a fall in blood pressure; patients should be advised to consult with the physician.

Hyperkalemia: Patients should be told not to use salt substitutes containing potassium without consulting their physician

Neutropenia. Patients should be told to report promptly any indication of infection (e.g., sore throat, fever) which may be a sign of neutropenia

NOTE. As with many other drugs, certain advice to patients being treated with enalapril is warranted. This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects.

Drug Interactions:

Hypotension. Patients on Diuretic Therapy: Patients on diuretics and especially those in whom diuretic therapy was recently instituted may occasionally experience an excessive reduction of blood pressure after initiation of therapy with enalapril. The possibility of hypotensive effects with enalapril can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with enalapril. If it is necessary to continue the diuretic, provide close medical supervision after the initial dose for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and DOSAGE AND ADMINISTRATION.)

Agents Causing Renin Release: The antihypertensive effect of VASOTEC is augmented by antihypertensive agents that cause renin release (e.g., diuretics).

Other Cardiovascular Agents: VASOTEC has been used concomitantly with beta-adrenergic-blocking agents, methyldopa, nitrates, calcium-blocking agents, hydralazine, prazosin, and digoxin without evidence of clinically significant adverse interactions

Agents Increasing Serum Potassium. VASOTEC attenuates potassium loss caused by thiazide-type diuretics. Potassium-sparing diuretics (e.g., spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium. Therefore, if concomitant use of these agents is indicated because of demonstrated hypokalemia, they should be used with caution and with frequent monitoring of serum potassium. Potassium-sparing agents should generally not be used in patients with heart failure receiving VASOTEC

Lithium. A few cases of lithium toxicity have been reported in patients receiving concomitant VASOTEC and lithium and were reversible upon discontinuation of both drugs. Although a causal relationship has not been established, it is recommended that caution be exercised when lithium is used concomitantly with VASOTEC and serum lithium levels should be monitored frequently.

Pregnancy—Category C: There was no fetotoxicity or teratogenicity in rats treated with up to 200 mg/kg/day of enalapril (333 times the maximum human dose). Fetotoxicity, expressed as a decrease in average fetal weight, occurred in rats given 1200 mg/kg/day of enalapril but did not occur when these animals were supplemented with saline. Enalapril was not teratogenic in rabbits. However, maternal and fetal toxicity occurred in some rabbits at doses of 1 mg/kg/day or more. Saline supplementation prevented the maternal and fetal toxicity seen at doses of 3 and 10 mg/kg/day, but not at 30 mg/kg/day (50 times the maximum human dose).

Radioactivity was found to cross the placenta following administration of labeled enalapril to pregnant hamsters

There are no adequate and well-controlled studies in pregnant women. VASOTEC® (Enalapril Maleate, MSD) should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers: Milk in lactating rats contains radioactivity following administration of ^{14}C enalapril maleate. It is not known whether this drug is secreted in human milk. Because many drugs are secreted in human milk, caution should be exercised when VASOTEC is given to a nursing mother.

Pediatric Use: Safety and effectiveness in children have not been established

Adverse Reactions: VASOTEC has been evaluated for safety in more than 10,000 patients, including over 1000 patients treated for one year or more. VASOTEC has been found to be generally well tolerated in controlled clinical trials involving 2987 patients.

Hypertension: The most frequent clinical adverse experiences in controlled trials were: headache (5.2%), dizziness (4.3%), and fatigue (3%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in controlled clinical trials were: diarrhea (1.4%), nausea (1.4%), rash (1.4%), cough (1.3%), orthostatic effects (1.2%), and asthenia (1.1%).

Heart Failure: The most frequent clinical adverse experiences in both controlled and uncontrolled trials were: dizziness (7.9%), hypotension (6.7%), orthostatic effects (2.2%), syncope (2.2%), cough (2.2%), chest pain (2.1%), and diarrhea (2.1%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in both controlled and uncontrolled clinical trials were: fatigue (1.8%), headache (1.8%), abdominal pain (1.6%), asthenia (1.6%), orthostatic hypotension (1.6%), vertigo (1.6%), angina pectoris (1.5%), nausea (1.3%), vomiting (1.3%), bronchitis (1.3%), dyspnea (1.3%), urinary tract infection (1.3%), rash (1.3%), and myocardial infarction (1.2%).

Other serious clinical adverse experiences occurring since the drug was marketed or adverse experiences occurring in 0.5% to 1% of patients with hypertension or heart failure in clinical trials in order of decreasing severity within each category.

Cardiovascular. Myocardial infarction or cerebrovascular accident, possibly secondary to excessive hypotension in high-risk patients (see WARNINGS, *Hypotension*); cardiac arrest, pulmonary embolism and infarction, rhythm disturbances; atrial fibrillation; palpitation.

Digestive: Ileus, pancreatitis, hepatitis or cholestatic jaundice, melena, anorexia, dyspepsia, constipation, glossitis

Nervous/Psychiatric. Depression, confusion, ataxia, somnolence, insomnia, nervousness, paresthesia

Urogenital: Renal failure, oliguria, renal dysfunction (see PRECAUTIONS and DOSAGE AND ADMINISTRATION), prostate hypertrophy.

Respiratory: Bronchospasm, rhinorrhea, asthma, upper respiratory infection

Skin: Herpes zoster, pruritus, alopecia, flushing, photosensitivity.

Other: Muscle cramps, hyperhidrosis, impotence, blurred vision, taste alteration, tinnitus.

A symptom complex has been reported which may include fever, myalgia, and arthralgia, an elevated erythrocyte sedimentation rate may be present. Rash or other dermatologic manifestations may occur. These symptoms have disappeared after discontinuation of therapy.

Angioedema: Angioedema has been reported in patients receiving VASOTEC (0.2%). Angioedema associated with laryngeal edema may be fatal. If angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with VASOTEC should be discontinued and appropriate therapy instituted immediately. (See WARNINGS.)

Hypotension: In the hypertensive patients, hypotension occurred in 0.9% and syncope occurred in 0.5% of patients following the initial dose or during extended therapy. Hypotension or syncope was a cause for discontinuation of therapy in 0.1% of hypertensive patients. In heart failure patients, hypotension occurred in 6.7% and syncope occurred in 2.2% of patients. Hypotension or syncope was a cause for discontinuation of therapy in 1.9% of patients with heart failure. (See WARNINGS.)

Clinical Laboratory Test Findings.

Serum Electrolytes: Hyperkalemia (see PRECAUTIONS), hyponatremia

Creatinine, Blood Urea Nitrogen: In controlled clinical trials, minor increases in blood urea nitrogen and serum creatinine, reversible upon discontinuation of therapy, were observed in about 0.2% of patients with essential hypertension treated with VASOTEC alone. Increases are more likely to occur in patients receiving concomitant diuretics or in patients with renal artery stenosis. (See PRECAUTIONS.) In patients with heart failure who were also receiving diuretics with or without digitalis, increases in blood urea nitrogen or serum creatinine, usually reversible upon discontinuation of VASOTEC and/or other concomitant diuretic therapy, were observed in about 11% of patients. Increases in blood urea nitrogen or creatinine were a cause for discontinuation in 1.2% of patients.

Hemoglobin and Hematocrit: Small decreases in hemoglobin and hematocrit (mean decreases of approximately 0.3 g % and 1.0 vol %, respectively) occur frequently in either hypertension or heart failure patients treated with VASOTEC but are rarely of clinical importance unless another cause of anemia coexists. In clinical trials, less than 0.1% of patients discontinued therapy due to anemia

Other (Causal Relationship Unknown): In marketing experience, rare cases of neutropenia, thrombocytopenia, and bone marrow depression have been reported.

Liver Function Tests: Elevations of liver enzymes and/or serum bilirubin have occurred

Dosage and Administration: *Hypertension:* In patients who are currently being treated with a diuretic, symptomatic hypotension occasionally may occur following the initial dose of VASOTEC. The diuretic should, if possible, be discontinued for two to three days before beginning therapy with VASOTEC to reduce the likelihood of hypotension. (See WARNINGS.) If the patient's blood pressure is not controlled with VASOTEC alone, diuretic therapy may be resumed

If the diuretic cannot be discontinued, an initial dose of 2.5 mg should be used under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions.*)

The recommended initial dose in patients not on diuretics is 5 mg once a day. Dosage should be adjusted according to blood pressure response. The usual dosage range is 10 to 40 mg per day administered in a single dose or in two divided doses. In some patients treated once daily, the antihypertensive effect may diminish toward the end of the dosing interval. In such patients, an increase in dosage or twice-daily administration should be considered. If blood pressure is not controlled with VASOTEC alone, a diuretic may be added

Concomitant administration of VASOTEC with potassium supplements, potassium salt substitutes, or potassium-sparing diuretics may lead to increases of serum potassium (see PRECAUTIONS).

Dosage Adjustment in Hypertensive Patients with Renal Impairment. The usual dose of enalapril is recommended for patients with a creatinine clearance >30 mL/min (serum creatinine of up to approximately 3 mg/dL). For patients with creatinine clearance ≤30 mL/min (serum creatinine ≥3 mg/dL), the first dose is 2.5 mg once daily. The dosage may be titrated upward until blood pressure is controlled or to a maximum of 40 mg daily.

Heart Failure: VASOTEC is indicated as adjunctive therapy with diuretics and digitalis. The recommended starting dose is 2.5 mg once or twice daily. After the initial dose of VASOTEC, the patient should be observed under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions.*) If possible, the dose of the diuretic should be reduced, which may diminish the likelihood of hypotension. The appearance of hypotension after the initial dose of VASOTEC does not preclude subsequent careful dose titration with the drug, following effective management of the hypotension. The usual therapeutic dosing range for the treatment of heart failure is 5 to 20 mg daily given in two divided doses. The maximum daily dose is 40 mg. Once-daily dosing has been effective in a controlled study, but nearly all patients in this study were given 40 mg, the maximum recommended daily dose, and there has been much more experience with twice-daily dosing. In addition, in a placebo-controlled study which demonstrated reduced mortality in patients with severe heart failure (NYHA Class IV), patients were treated with 2.5 to 40 mg per day of VASOTEC, almost always administered in two divided doses. (See CLINICAL PHARMACOLOGY, *Pharmacodynamics and Clinical Effects.*) Dosage may be adjusted depending upon clinical or hemodynamic response. (See WARNINGS.)

Dosage Adjustment in Heart Failure Patients with Renal Impairment or Hyponatremia. In heart failure patients with hyponatremia (serum sodium <130 mEq/L) or with serum creatinine >1.6 mg/dL, therapy should be initiated at 2.5 mg daily under close medical supervision. (See DOSAGE AND ADMINISTRATION, *Heart Failure*, WARNINGS, and PRECAUTIONS, *Drug Interactions.*) The dose may be increased to 2.5 mg b.i.d., then 5 mg b.i.d. and higher as needed, usually at intervals of four days or more, if at the time of dosage adjustment there is not excessive hypotension or significant deterioration of renal function. The maximum daily dose is 40 mg.

For more detailed information, consult your MSD representative or see Prescribing Information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, PA 19486. J6VS18R(815)

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News from the Inuit Circumpolar Conference

Briefing Notes Regarding International Dimensions of Arctic Toxic Contaminants

March 1, 1989

Evidence is emerging to the effect that the arctic has suffered widespread contamination with a variety of toxic chemicals, including PCBs, DDTs, and many others.

Chemical and Biological Aspects

These compounds are long-chain complex organic molecules. One of the most significant features of these compounds on entering living organisms is their affinity for fatty tissues. Contamination of living organisms is cumulative because there are no decontamination mechanisms and the chemicals continue to accumulate in the body, subsequent exposures resulting in higher and higher loads of chemicals. This process is known as biomagnification. Some of this load is shed by mammalian females when they carry developing embryos and these chemicals enter the developing fetus, or during lactation through the excretion of these chemicals in mothers milk.

The dangers associated with DDT compounds were eloquently described by Rachel Carson in her book "Silent Spring," which inspired the American environmental movement to bring about the ban on their use in the United States. Concerns about the impact of DDTs on eggshell thickness and reproductive success of falcons and eagles led European countries to eliminate their use, but unfortunately use in other regions of the world continues.

PCBs have been believed to be relatively harmless for some time. Recent studies, however, have begun to link PCBs to reproductive failure in various species, and to learning and behavioral dysfunction and immunesuppression in human beings. Very recently it has been suggested that the most critical exposure for humans is exposure of the fetus in utero to PCBs which cross the placental barrier.

Sources of Toxic Contaminants Found in the Arctic Environment

PCBs were used in capacitors in electrical generators and as fire-retardants. DDTs and related chemicals were designed as insecticides. These chemicals have been used extensively at different times throughout the developed and developing world.

With respect to the arctic, while government has known about the presence of PCBs in polar bears since 1985, it was generally thought that the sources of such contamination must be local point sources related to previous military and industrial activities. For example, many abandoned DEW-line sites were found to be contaminated with PCBs and a thorough clean-up was to be completed under the supervision of DIAND. This clean-up was conducted, but the extent to which sites remained contaminated, either because the clean-up was inadequate or because contaminated sites were missed, is not known. For example, Canadian and U.S. military used PCBs, as did the Northern Canada Power Corporation, and some materials from these operations were disposed of in community dumps. The PCB clean-up operation addressed known military sites but did not survey community dumps. Also PCB contaminated materials found their way from abandoned sites and community dumps into communities where they appear to have been recycled for a number of purposes.

More recently it has been suggested that atmospheric transfer of contaminants is occurring on a large scale and that the greatest threat to the arctic environment and peoples is due to deposition from the atmosphere. Atmospheric deposition from sources abroad, coupled with the bioaccumulation of these toxic chemicals, makes immediate international action on these issues imperative. Unless these toxic chemicals are controlled at their sources, they will continue to be transported to the arctic and will continue to accumulate in the living organisms, including people. As new research suggests that higher levels of toxics in the body are associated with higher risks (particularly with respect to offspring), additional exposure to these toxics leading to increased levels must be eliminated as quickly as possible.

Resolution on Contamination of the Arctic by Toxic Chemicals

Be it Resolved

THAT ICC call on its affilitated organizations, including authorities, to adopt resolutions calling for immediate government action with respect to an international agreement to protect the arctic environment and its peoples from toxic contamination originating in industrialized regions; for the release of all available information to Inuit; and for direct Inuit involvement through the Inuit Circumpolar Conference in any research initiatives undertaken with respect to this issue;

THAT ICC take the lead in coordinating an Inuit call for international action on toxic contamination of the arctic on a circumpolar basis, together with the necessary lobbying initiatives;

THAT ICC call on the governments of Canada, Greenland/Denmark, and Alaska/USA to make resources available to the ICC for research, coordination and communications, including all information available on this issue;

THAT the ICC General Assembly to be held this summer give consideration to the adoption of a resolution calling on circumpolar governments to give highest priority to an international agreement dealing with toxic contamination of the arctic;

THAT adequate studies be conducted by the appropriate governments and other agencies to monitor effects on Inuit and the living resources of the arctic, and that the Inuit Circumpolar Conference be directly involved in an advisory capacity with respect to such research, through participation in a steering committee or other appropriate mechanism.

Resolution 86-07

Environmental Causes of Health Problems in the Arctic Regions

WHEREAS, old military installations, White Alice sites and DEW Line sites cause environmental and health problems in the Arctic Regions; and

WHEREAS, contamination is causing cancer and environmental problems for the Inuit of the circumpolar regions;

NOW THEREFORE BE IT RESOLVED THAT the Inuit Circumpolar Conference demands that the governments of the United States, Canada and Denmark initiate clean-up of all the old military installations, army sites, White Alice sites, and DEW Line sites as soon as possible; and

BE IT FURTHER RESOLVED THAT the Inuit Circumpolar Conference do an extensive survey on the effects of military installations on the health and well-being of the Inuit in the circumpolar regions.

Resolution 86-17

Arctic Haze

WHEREAS, Arctic air masses have been scientifically proven to be polluted; and

WHEREAS, Arctic air pollution is derived mainly from industrial sources outside the geographical regions of the Arctic and carried into the Arctic by general circulation of the atmosphere; and

WHEREAS, the Arctic haze is thickest in the winter months when polar air masses are in a stagnant condition; and

WHEREAS, there is a need to have on-going coordinated scientific research to determine the consequences of Arctic air pollution, especially its impact on regional and global climate, human health, and sensitive ecological systems;

NOW THEREFORE BE IT RESOLVED THAT the Inuit Circumpolar Conference encourage Arctic-wide systematic sampling of air to assess the present and future status of Arctic air-shed; and

BE IT FURTHER RESOLVED THAT the Inuit Circumpolar Conference encourages the cooperation of Nations and their respective scientific talent from universities, industries, and government agencies in solving the general problem of air pollution.

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President's Page

Not long ago, while on a trip outside to get away from the Alaskan winter, I awoke one morning to find the Alaskan winter had come with me. It was snowing heavily. I made a pot of coffee, decided there was no reason to shave and get dressed as I was not going anywhere, and settled in for a few hours of reading. A day or so before, while poking around in a used book store, I had found a Joan Didion novel which I had not read before. And since no one writes better about the 1960s than she does, I was glad for the excuse to stay in and read.

The novel, *Play It as It Lays*, is a compelling story. It follows a woman in her early thirties through several critical months of her life in Los Angeles in the late 1960s and is written with the vividness and insight that are typical of the author.

But I am not promoting book sales for Ms. Didion. The reason I mention the book is that it brought back into focus a medical problem I have not had to deal with in years; the fear and helplessness of the woman pregnant with a child she feels she cannot have and who resorts to an illegal abortion.

The story follows a young woman as she discovers that she is pregnant, agonizes over what to do, turns to her estranged husband and her friends for help; and, finding none, endures the back alley furtiveness of looking for an abortionist and faces the guilt about the abortion—alone. This is the stuff of soap operas, but the book is powerfully written, without self pity, and is in no way maudlin.

Reading this at the time when there is much support for again making abortion illegal adds the impact of immediacy. We may soon be back in the situation described by this novel, placed there either by judicial fiat, or less probably by constitutional amendment.

Certainly legal abortion is not the answer to the problem of unwanted pregnancy. The woman who chooses to terminate her pregnancy is not relieved of fear and guilt simply because the abortion is sanctioned by statute. She is only relieved of the unnecessary aggravation of these emotions which resorting to an illegal, and unnecessarily dangerous, procedure brings about. A woman whose ethical and moral standards allow her the option of terminating her pregnancy, should not be denied the right to do so even though there are many among us who cannot agree with her decision.

Irvin A. Rothrock, M.D.
President
Alaska State Medical Association

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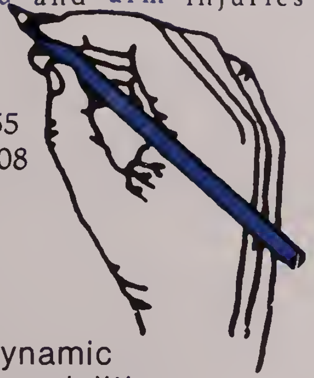

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History of Medicine in Alaska

HARRIET CROCKER JACKSON SCHIRMER, M.D.

To understand Harriet Jackson Schirmer, M.D., we must begin with Mildred Wicker, her mother, who was a doctor's daughter.

At twelve Mildred was doing drop ether for tonsillectomies when the specialist came to town. Afterwards, she was left to watch the patients in the recovery room. When Mildred decided to go to medical school, her grandmother was asked if it would be all right. She replied: "Yes, if she behaves like a lady." Harriet's mother went to Cornell Medical School. During the flu epidemic in 1918, she was put in charge of a ward even though it was only her first month in school. Mildred Wicker became a pediatrician.

She married Myron Jackson who was a general practitioner and surgeon.

Theodore Roosevelt's daughter had married a doctor, Richard Derby. Dr. Myron Jackson went to Oyster Bay to assist the son-in-law. Here Harriet was born. Her parents had offices in their home. Eventually, Harriet, the oldest, found herself assisting her father when patients came after hours.

Hers was a pleasant life with good schools and sailing boats.

At sixteen, she entered the University of Michigan for undergraduate school. She worked summers in a hospital. Her duties ranged from being an aid in the operating room, cleaning labs, and drawing blood to serving as an orderly in the X-ray department where she took EKGs and developed them in the darkroom. From these early days, she found chest x-rays the most difficult.

In another summer, she worked at the Carnegie Foundation Genetics Laboratory. After checking 1600 mice daily for pregnancy or liver tumors, she discovered that she didn't want to do research.

She entered the New York University Medical School. After the first year, she went to Europe attending the Olympic sailing at Torquay, England. Also she did much bicycling. A friend fractured her skull in Stratford-on-Avon. Because of the recently established National Health Service, she was not allowed to pay for anything.

However, Harriet's subsequent summers were business as usual. She reviewed autopsy reports at Bellevue Hospital for an urologist which was dull and then became an orthopedics extern.

Harriet Jackson interned at Meadowbrook Hospital in Nassau County, N.Y. While rotating through obstetrics, surgery, medicine, and psychiatry, she especially remembers being in the contagious diseases for four months with 250 polio patients.

Her two-year residency was at the University of Colorado Hospital where Helen Whaley was then chief pediatrics resident; John Finger, an intern; and Gloria Parks a medical student.

Because of "galloping suburbia" and the growing specialization of medicine, she knew, even before medical school, that she intended to go to Alaska.

Everywhere she went, she was able to find training for special procedures she though she might need in Alaska. While she was in medicine at Denver General, she was placed in charge of the tuberculosis wards at the Denver General Hospital and then went to a remote area in southern Colorado to a small hospital. Before going she had learned she might have to do her own X-rays and autopsies. So, she prepared in Denver by studying gross pathology.

Harriet did come to Alaska in 1954 as a civil servant physician for the Alaska Native Health Service. She was sent to Bethel. This was the beginning of the 11 years she spent on the Kuskokwim. The first three, she was in charge of the almost brand new hospital. The hospital had been a quonset hut with 10 beds. The new building of 50 beds had opened just before she got there. One of her first chores was to requisition supplies, both food and medicine, for the entire year.

In the pharmacy she found drugs that her parents called "historical medications," because her grandfather had used them.

The overwhelming problem was tuberculosis. The first year that Harriet Jackson was in Bethel, 90% of the children under two years tested positive for TB; even those under three months who usually aren't tested for it. The Bureau of Indian Affairs policy was to take only primary or minimal disease patients to the hospital so that too many would not die, thereby giving a negative impression of hospitalization. Soon there were 300 beds in Seattle where the patients were sent, and a waiting list of two years disappeared in about one year.

Harriet took a portable X-ray machine into the villages, rounding everyone up. It was impossible to prevent the disease when seven people were living in one small room. The choices were tragic—allow a newborn to get TB from its ill mother or die in isolation from her.

Dr. Beryl Michaelson pioneered giving streptomycin and isoniazide in the villages. Harriet was able to expand this program. By 1965, only about 1% of preschool children in Bethel were to skin test TB positive. In balance, she remembers one man who started working at the hospital after being incapacitated for 17 years by tuberculosis. He became a reliable long term employee.

She was frustrated by the Native attitudes which often she learned by chance. Undoubtedly, the Native, who was trying to comply with a misunderstood concept, was equally frustrated. It was only by accident that she learned that the Native held that if you took "Gussack" medicine, you couldn't eat Eskimo food. She learned this from a woman from Oscarville who told her that she had taken the medicine as long as she could but that she thought she would die of hunger. Harriet sent nurses to persuade the Natives that fish and mink stew, etc., were acceptable while taking Gussack medicine. Apparently, this misconception had developed from a well-intended nurse some 15 years earlier who had in her pharmacy only cod liver oil and castor oil and had warned everyone not to eat native foods while taking these medications.

Harriet attempted to learn Yupik but got off to a poor start, using the word for "bad," thinking it meant "good."

Once she took a 14-year old girl to Tuntuliak as a guide-interpreter. The girl would not go into the sod house of a bachelor. The man was suffering from orchitis. Even without the help of an interpreter, Harriet could understand and used the then popular cortisone treatment which relieved pain and swelling. However, she never knew whether he had been left with infertility.

In 1956, on a field trip, she found herself at Holy Cross where she spent the night in the Bishop's room in his absence. The villagers persuaded her to go to a remote village where there was no doctor. She was exhilarated by the experience of traveling with five dog sleds running parallel. The group stopped for an al fresco lunch at a temporarily vacated baraberra (sod hut). In order to have room to make tea, they removed a huge kayak. Before they left, everything was carefully replaced.

Harriet was moved by visiting an old, blind woman. It was touching that, having asked for water, before drinking it, the 94-year old woman crossed herself and gave thanks.

Harriet did see the sick in the village. But she was most impressed with the school teacher, a Native, who taught English to Eskimo children. Within three days the children had mastered complicated concepts in English. She enjoyed the ease with which they grasped weather terms such as visibility and precipitation.

For two years, she was in charge of a project on chronic otitis media for the Children's Bureau. This was a big problem in the villages. Ear infections in children often were allowed to go on for two to six months before being seen, and by then they had become chronic infections. She was assisted by two part-time nurses and an educator. The major thrust was education, explaining that an ear treated when the infection first began could be cured. At this time sulfonamide was the treatment, but later ampicillin was to work more effectively.

Harriet Jackson went into private practice in Bethel competing, as she puts it, with the government. She was to have a large obstetrics practice among the non-Natives since they were not covered by the PHS. She delivered babies in her office.

She was amused to note that while all the women were treated in the hospital for gonorrhea, the men came to her office. The explanation was simple. The men, who were employed, could not afford to wait. It was to their advantage to pay her.

She was taught to do eye refractions by Joe Sheldon, M.D.. She would send the prescriptions to Anchorage opticians. This was the way she managed to maintain her practice.

When Donald Schirmer came to Bethel in charge of the White Alice System, things changed. He claimed to have a broken toe which needed X-raying. Having thus met, he tested Harriet further by taking her flying. Then they were married.

After several long cold winters, Don became tired of the problems of getting crews to the site. After 40 years of living in Oyster Bay, Harriet's mother had sold her house, intending to return to her hometown to retire with a school teacher friend. However, the friend developed cancer. Harriet's mother had continued to do pediatric consultations until her income was smaller than the malpractice insurance premiums. Mildred Jackson was 78 years old at this time.

The Schirmers, including son Jack, moved to upstate New York where Mildred Jackson now retired. Don farmed and worked for an Apollo program. Harriet practiced, but was 25 miles from the nearest hospital. They were to remain in Livonia for five years.

Don made a trip to Alaska to find a place where both of them could find things to do. This turned out to be Wrangall. For ten years, he was the head electrician at the sawmill and then, after working on the Typee hydro project, he retired.

Harriet found that people came to her for small things, but not important ones. So she purchased a boat, put an office on it, and for ten years, every summer, visited the logging camps and the fishing villages. The logging camps were temporary communities of approximately 300 people, some with schools and houses built on rafts.

The fishing villages were mainly non-Native. There were no historic Native villages closer than Klawock.

In spite of the whales and the birds, they sold the boat. Harriet has retired, which seems hard to believe. Active people cannot avoid finding things to do. It will be fascinating to see what is next for Harriet Crocker Jackson Schirmer.

Gwynneth Gminder Wilson
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My Turn

The "New" Thermal Unit

by Robert E. Mallin, M.D.[1]

It has been almost exactly ten years since I wrote an article for this Journal; it was about the new Thermal Unit to be opened at Providence hospital. A lot has happened since then.

Providence, itself, has grown from the designation of "The hospital" (south tower) and "The new part" (north tower), to the multi-towered, turreted, departmentalized, structured, organized, administrated, and landscaped project of the 90s. The original thermal people have either passed on or gotten older. Don Addington and Emmett Bolt are gone. I was much thinner with more hair. Paul Steer, M.D. was actually almost a red head, while of course George Stewart, M.D. and Bill Mills, M.D. haven't aged a bit (just ask them). Archbishop Hearly, Sister Clair and the Pargetters who took part in the earlier opening as prime movers are still, more or less in place, though Mr. Camosso is not. Original major funding was from Mr. and Mrs. Ray Kroc (McDonalds), Jim Laniers' Iditarod-A-Thon, and the Fire Fighters. As you may remember, the old surgical ICU had been remodeled, and we were underway.

Hundreds of patients have been treated with hot and cold injuries since that time. Many languages have been spoken, and various degrees of severity and injury have been seen. Mountain climbers from abroad, cattle barons on hunting trips, frozen foreign sea men, and others. In short, people from all areas and occupations of Alaska and the whole world have passed through our portals. (How do you say, "Take a deep breath" in Japanese?) However, with the changes in population, economy, and state of the art technology, not to mention Providence's building program, the Thermal Unit must move on. We are moving now to the old CCU of the North Tower. It is refurbished with better rooms than the current thermal unit, has much better equipment, and certainly has a more centralized location.

Concepts have changed from the old idea of a centralized Thermal Unit to a more or less "hub and satellite system." The Thermal Unit itself will be the Thermal Intensive Care Unit, the hub of critical new patients and administrative work, while the lesser injured and healing patients can be promoted to the general floors. Part of the proposal is for a group of three West rooms to be used for the unique requirements of the frost bitten and hypothermic patients, as well as a place to house Dr. Mills' on-going research projects. The nursing concept has also changed. The primary goals are still optimum patient care. There will be one person in overall charge of the thermal service, a person responsible for the unit patients, and at least one person responsible for outlying and outpatients. For practical purposes, this means we can call one person and get a full report on all of our patients. This way, hopefully, we can avoid the all too familiar "It's not my patient, and Jane is on a break." The extended team approach, of course, continues. We will have continuity of nursing care, more physical therapy, occupational therapy, rehabilitation, and ancillary support. David Brauner, M.D., is essentially our pediatric thermal specialist, while Internal Medicine Associates continues to provide maximum medical support. Fred Hood, M.D., George Siegfried, M.D., and I, divide the surgical call.

Over the past ten years major changes in treatment have come about. These include primary excision and closure of burn wounds, often requiring two to three surgeries within the first ten days to two weeks. Parentral nutrition has come into its own, as well as the concept of rehabilitation and reconstruction to start during the first emergency room visit. Aggressive respiratory treatment as well as P.T., O.T., and splinting have greatly improved the quality as well as the quantity of life. No longer is a burn unit content with saving a life unless the person can be reintegrated into society. Happily, our infectious disease control procedures have been modified in the new unit with the whole central area being neutral. That is, no special garments to be worn, and individual sterilization procedures are determined for the individual patient rooms.

We are quite proud of our new unit and consider this part of the modernization program that has taken place in medicine in general, and Providence Hospital in particular. Please come and take a look, somebody will always be happy to answer your questions and show you around.

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Editorial

The health status of Alaska's environment moved to center stage when we learned of the largest oil spill in U.S. history that occurred in Prince William Sound the night of March 24. Crude oil had been shipped successfully by tanker from Valdez Harbor for more than 12 years, although several major disasters were narrowly averted in the past. Catastrophe struck in perfect weather—calm seas, no wind, and clear visibility. The full impact of the spill is unknown at this time.

By the second day after the ship ran aground, oil had spread to cover a huge area. By Monday, only two options appeared viable to clean up the massive amount of viscous, crude oil. One was to use chemicals to break up and disperse the oil, the other was to ignite the oil. Neither held much hope of success.

Unfortunately, as all too often the case, decisions on how to proceed had to be made on the spot, quickly, and without adequate knowledge, data, and experience. For example, the composition of the recommended chemical dispersant was proprietary, so an independent assessment of its potential toxicity could not be made. A decision to ignite the crude oil had to weigh the potential for adverse health effects from the smoke to residents of the villages and communities nearby and downwind of the spill site. How could anyone predict with certainty the amount of smoke, the speed and direction of the prevailing winds, and the amount of oil likely to burn? Did Robert Service write presciently, "It was icy cold, but the hot sweat rolled down my cheeks, and I don't know why; And the greasy smoke in an inky cloak went streaking down the sky." (The Cremation of Sam McGee)?

The shear magnitude of the potential environmental catastrophe all but pushed other matters aside. However, let us not forget other members of the cast of recent environmental incidents sharing the stage, all be it, now, at least temporarily, in lesser roles.

- The state is currently in the midst of negotiations with the companies engaged in mining and transporting lead and zinc ore about the environmental clean up in Skagway.
- The state recently settled a claim for more than $200,000 over indoor air pollution in a building in Anchorage. Many worker's compensation claims related to the building are pending, as well as several lawsuits.
- Residents of Crown Point are proceeding with a $86 million lawsuit over their exposure to chemicals released as a result of an accidentally initiated, uncontrolled chemical reaction in a tank car.
- Controversy surrounds the environmental impact statement (EIS) prepared in anticipation of proposed expansion of offshore dredging for gold near Nome in Norton Sound. The EIS raised concerns that mercury in the ocean bottom sediments might, if disturbed by the large scale proposed dredging operations, bioaccumulate and cause adverse health effects among people who consume seafood or marine mammals.
- Tetrachloroethylene (TCE), a chemical contaminant, was detected in the drinking water of several Girdwood residents.
- Concerns recently have been voiced about possible adverse health effects due to sulfur dioxide (SO2) in emissions from the Sitka pulp mill.
- Concerns about radon have increased our anxieties and made us increasingly suspicious of unseen, undetectable, physical and chemical threats in buildings in which we work and in our homes.
- Recent national publicity focused on "contamination" of apples by the pesticide, Alar. A publicity exclusive, gone bad, resulted in needless fear and anxiety.

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Critical to assessing these potential environmental threats is science. Scientists must provide accurate and factual information in a timely way. Considering that recent surveys show a disgraceful lack of science knowledge among high school graduates (for example, fewer than half of graduating seniors know that the country bordering the United States to the south is Mexico), this task is not easy. And of greatest importance, scientists must identify the limits of current knowledge so that clear distinctions can be made between science and belief.

Science relies on observations, measurements, and discoveries that are successfully tested again and again. The scientific method requires that scientists remain open-minded, willing to accept changes indicated by evidence, and willing to submit their theories to rational examination.

Scientists must be able to show where scientific knowledge (and consensus of evidence) stops, where hypotheses and theories end, and where beliefs, prejudice, politics, and value-laden judgments begin.

We have not served the public, and we have not served science well when we allow scientific knowledge to be misinterpreted, misrepresented, or distorted. We have not served science well when we do not define the distinctions between scientific knowledge and wishful thinking. We victimize the mother who, upon hearing national media coverage about Alar and apples, worries herself to death, knocks back a couple of drinks, smokes a few cigarettes, and drives at high speed without being buckled up to school to remove an apple from her child's lunchbox.

We need to use our available knowledge wisely. Experience is one thing we can't get for nothing. But, we have all too much experience with environmental disasters and policy failures. We must use our past experiences. We must combine our powerful scientific knowledge with our experience so that we can make wise decisions as individuals and as a society.

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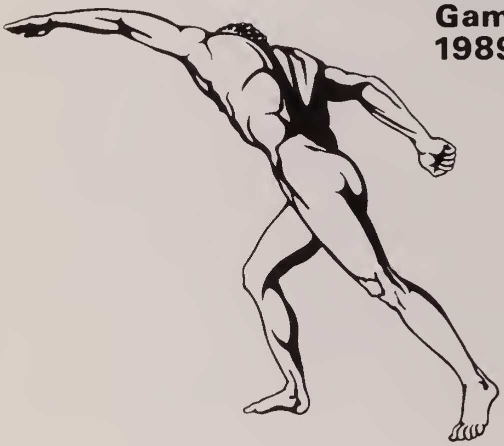

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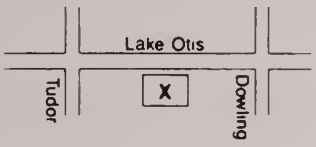

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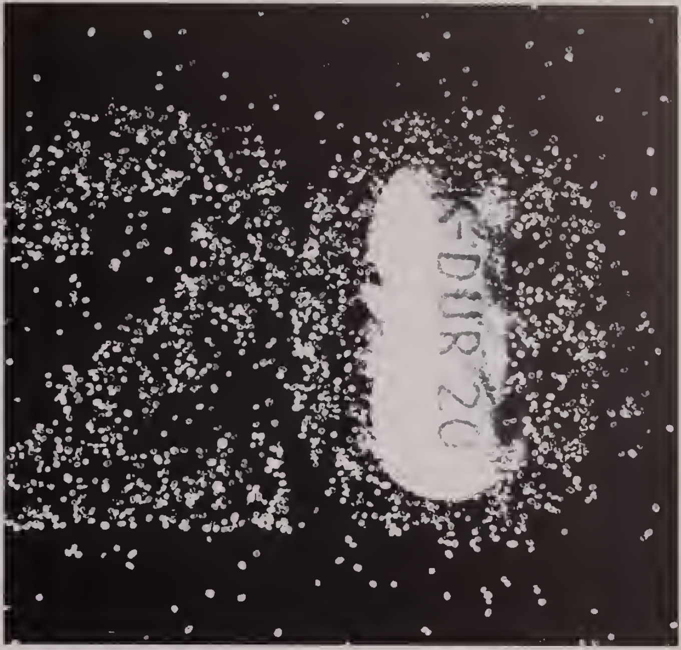

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1. For therapeutic use in patients with hypokalemia with or without metabolic alkalosis, in digitalis intoxication and in patients with hypokalemic familial periodic paralysis.

2. For the prevention of potassium depletion when the dietary intake is inadequate in the following conditions: Patients receiving digitalis and diuretics for congestive heart failure, hepatic cirrhosis with ascites, states of aldosterone excess with normal renal function, potassium-losing nephropathy, and with certain diarrheal states.

3 The use of potassium salts in patients receiving diuretics for uncomplicated essential hypertension is often unnecessary when such patients have a normal dietary pattern. Serum potassium should be checked periodically, however, and if hypokalemia occurs, dietary supplementation with potassium-containing foods may be adequate to control milder cases. In more severe cases supplementation with potassium salts may be indicated

CONTRAINDICATIONS: Potassium supplements are contraindicated in patients with hyperkalemia since a further increase in serum potassium concentration in such patients can produce cardiac arrest Hyperkalemia may complicate any of the following conditions: Chronic renal failure, systemic acidosis such as diabetic acidosis, acute dehydration, extensive tissue breakdown as in severe burns, adrenal insufficiency, or the administration of a potassium-sparing diuretic (e.g , spironolactone, triamterene).

Wax-matrix potassium chloride preparations have produced esophageal ulceration in certain cardiac patients with esophageal compression due to enlarged left atrium.

All solid dosage forms of potassium chloride supplements are contraindicated in any patient in whom there is cause for arrest or delay in tablet passage through the gastrointestinal tract. In these instances, potassium supplementation should be with a liquid preparation.

WARNINGS: **Hyperkalemia**—In patients with impaired mechanisms for excreting potassium, the administration of potassium salts can produce hyperkalemia and cardiac arrest. This occurs most commonly in patients given potassium by the intravenous route but may also occur in patients given potassium orally. Potentially fatal hyperkalemia can develop rapidly and be asymptomatic. The use of potassium salts in patients with chronic renal disease, or any other condition which impairs potassium excretion, requires particularly careful monitoring of the serum potassium concentration and appropriate dosage adjustment

Interaction with Potassium Sparing Diuretics—Hypokalemia should not be treated by the concomitant administration of potassium salts and a potassium-sparing diuretic (e.g., spironolactone or triamterene) since the simultaneous administration of these agents can produce severe hyperkalemia.

Gastrointestinal Lesions—Potassium chloride tablets have produced stenotic and/or ulcerative lesions of the small bowel and deaths. These lesions are caused by a high localized concentration of potassium ion in the region of a rapidly dissolving tablet, which injures the bowel wall and thereby produces obstruction, hemorrhage or perforation.

K-DUR tablets contain micro-crystalloids which disperse upon disintegration of the tablet. These micro-crystalloids are formulated to provide a controlled release of potassium chloride. The dispersibility of the micro-crystalloids and the controlled release of ions from them are intended to minimize the possibility of a high local concentration near the gastrointestinal mucosa and the ability of the KCl to cause stenosis or ulceration. Other means of accomplishing this (e.g., incorporation of potassium chloride into a wax matrix) have reduced the frequency of such lesions to less than one per 100,000 patient years (compared to 40–50 per 100,000 patient years with enteric-coated potassium chloride) but have not eliminated them. The frequency of GI lesions with K-DUR tablets is, at present, unknown. K-DUR tablets should be discontinued immediately and the possibility of bowel obstruction or perforation considered if severe vomiting, abdominal pain, distention, or gastrointestinal bleeding occurs

Metabolic Acidosis—Hypokalemia in patients with metabolic acidosis should be treated with an alkalinizing potassium salt such as potassium bicarbonate, potassium citrate, potassium acetate, or potassium gluconate.

PRECAUTIONS: The diagnosis of potassium depletion is ordinarily made by demonstrating hypokalemia in a patient with a clinical history suggesting some cause for potassium depletion. In interpreting the serum potassium level, the physician should bear in mind that acute alkalosis per se can produce hypokalemia in the absence of a deficit in total body potassium while acute acidosis per se can increase the serum potassium concentration into the normal range even in the presence of a reduced total body potassium. The treatment of potassium depletion, particularly in the presence of cardiac disease, renal disease, or acidosis requires careful attention to acid-base balance and appropriate monitoring of serum electrolytes, the electrocardiogram, and the clinical status of the patient

Laboratory Tests: Regular serum potassium determinations are recommended. In addition, during the treatment of potassium depletion, careful attention should be paid to acid-base balance, other serum electrolyte levels, the electrocardiogram, and the clinical status of the patient, particularly in the presence of cardiac disease, renal disease, or acidosis.

Drug Interactions: Potassium-sparing diuretics; see **WARNINGS**.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Long-term carcinogenicity studies in animals have not been performed

Pregnancy Category C: Animal reproduction studies have not been conducted with K-DUR. It is also not known whether K-OUR can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. K-OUR should be given to a pregnant woman only if clearly needed

Nursing Mothers: The normal potassium ion content of human milk is about 13 mEq per liter. Since oral potassium becomes part of the body potassium pool, so long as body potassium is not excessive, the contribution of potassium chloride supplementation should have little or no effect on the level in human milk

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS: One of the most severe adverse effects is hyperkalemia (see **CONTRAINDICATIONS, WARNINGS**, and **OVERDOSAGE**). There have also been reports of upper and lower gastrointestinal conditions including obstruction, bleeding, ulceration, and perforation (see **CONTRAINDICATIONS** and **WARNINGS**); other factors known to be associated with such conditions were present in many of these patients.

The most common adverse reactions to oral potassium salts are nausea, vomiting, abdominal discomfort, and diarrhea These symptoms are due to irritation of the gastrointestinal tract and are best managed by taking the dose with meals or reducing the dose

Skin rash has been reported rarely

OVERDOSAGE: The administration of oral potassium salts to persons with normal excretory mechanisms for potassium rarely causes serious hyperkalemia However, if excretory mechanisms are impaired or if potassium is administered too rapidly intravenously, potentially fatal hyperkalemia can result (see **CONTRAINDICATIONS** and **WARNINGS**). It is important to recognize that hyperkalemia is usually asymptomatic and may be manifested only by an increased serum potassium concentration and characteristic electrocardiographic changes (peaking of T-waves, loss of P-waves, depression of S-T segment, and prolongation of the OT-interval). Late manifestations include muscle-paralysis and cardiovascular collapse from cardiac arrest.

Treatment measures for hyperkalemia include the following

1 Elimination of foods and medications containing potassium and of potassium-sparing diuretics.

2 Intravenous administration of 300 to 500 ml/hr of 10% dextrose solution containing 10–20 units of insulin per 1,000 ml

3 Correction of acidosis, if present, with intravenous sodium bicarbonate.

4 Use of exchange resins, hemodialysis, or peritoneal dialysis

In treating hyperkalemia, it should be recalled that in patients who have been stabilized on digitalis, too rapid a lowering of the serum potassium concentration can produce digitalis toxicity

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BRIEF SUMMARY

CONTRAINDICATIONS

There are no known contraindications to the use of sucralfate.

PRECAUTIONS

Duodenal ulcer is a chronic, recurrent disease. While short-term treatment with sucralfate can result in complete healing of the ulcer, a successful course of treatment with sucralfate should not be expected to alter the post-healing frequency or severity of duodenal ulceration.

Drug Interactions: Animal studies have shown that simultaneous administration of CARAFATE (sucralfate) with tetracycline, phenytoin, digoxin, or cimetidine will result in a statistically significant reduction in the bioavailability of these agents. The bioavailability of these agents may be restored simply by separating the administration of these agents from that of CARAFATE by two hours. This interaction appears to be nonsystemic in origin, presumably resulting from these agents being bound by CARAFATE in the gastrointestinal tract. The clinical significance of these animal studies is yet to be defined. However, because of the potential of CARAFATE to alter the absorption of some drugs from the gastrointestinal tract, the separate administration of CARAFATE from that of other agents should be considered when alterations in bioavailability are felt to be critical for concomitantly administered drugs.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Chronic oral toxicity studies of 24 months' duration were conducted in mice and rats at doses up to 1 gm/kg (12 times the human dose). There was no evidence of drug-related tumorigenicity. A reproduction study in rats at doses up to 38 times the human dose did not reveal any indication of fertility impairment. Mutagenicity studies were not conducted.

Pregnancy: Teratogenic effects. Pregnancy Category B. Teratogenicity studies have been performed in mice, rats, and rabbits at doses up to 50 times the human dose and have revealed no evidence of harm to the fetus due to sucralfate. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when sucralfate is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Adverse reactions to sucralfate in clinical trials were minor and only rarely led to discontinuation of the drug. In studies involving over 2,500 patients treated with sucralfate, adverse effects were reported in 121 (4.7%).

Constipation was the most frequent complaint (2.2%). Other adverse effects, reported in no more than one of every 350 patients, were diarrhea, nausea, gastric discomfort, indigestion, dry mouth, rash, pruritus, back pain, dizziness, sleepiness, and vertigo.

OVERDOSAGE

There is no experience in humans with overdosage. Acute oral toxicity studies in animals, however, using doses up to 12 gm/kg body weight, could not find a lethal dose. Risks associated with overdosage should, therefore, be minimal.

DOSAGE AND ADMINISTRATION

The recommended adult oral dosage for duodenal ulcer is 1 gm four times a day on an empty stomach.

Antacids may be prescribed as needed for relief of pain but should not be taken within one-half hour before or after sucralfate.

While healing with sucralfate may occur during the first week or two, treatment should be continued for 4 to 8 weeks unless healing has been demonstrated by x-ray or endoscopic examination.

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1. Eliakim R, Ophir M, Rachmilewitz D: *J Clin Gastroenterol* 1987;9(4):395-399.

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Captain, U.S. Army Reserve.

EDUCATION University of Southern California, B.S.; University of California School of Medicine.

RESIDENCY Harbor General Hospital—UCLA Medical Center.

HOSPITAL AFFILIATIONS St. Luke Hospital; Huntington Memorial Hospital, Pasadena, California; Traumatologist, Arcadia Methodist Hospital, Arcadia, California.

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Emerging Public Health Issues in Alaska: Occupational and Environmental Health

by Lawrence D. Weiss, Ph.D., M.S.[1]
John M. Booker, Ph.D.[2]
David Wigglesworth[3]

ABSTRACT

The principal purpose of this article is to examine selected Alaskan occupational and environmental health problems and associated issues. Specifically, we focus on two traditional areas of interest in occupational and environmental health:

1) Job-related injuries, illnesses, and fatalities, and
2) accidental spills and uncontrolled waste sites involving hazardous materials.

In 1986 the Alaska Department of Labor reported 13,154 recordable occupational injuries and illnesses among workers in the private sector. The injury rate for private sector workers was 10.2 cases per 100, compared to a rate of 7.9 for the U.S. private sector as a whole. There are approximately 550 known hazardous waste sites in Alaska identified by military, other federal, or state sources. One study documents 1,330 hazardous materials incidents in Alaska during a one year period. Effective public health policy for these occupational and environmental health hazards requires the development of an adequate database and a specific plan of action for the future.

> "I am very concerned with the number of fatalities occurring in Alaska. During 1985 and 1986 there were 77 worker fatalities, not counting heart attack cases.
>
> For the first nine months of 1987, Alaska exceeded the number of workers deaths in 1986. According to a recent 1987 NIOSH study, Alaska has the second highest fatal work accident rate in the nation, with a rate nearly four times that of the U.S. average.
>
> I am especially worried with what is happening in Alaska's logging industry. We have already had nine fatalities this year alone."
>
> Jim Sampson
> Commissioner of Labor
> October 1987 (1)

INTRODUCTION

The principal purpose of this article is to examine selected Alaskan occupational and environmental health problems and associated issues. Specifically, we focus on two traditional areas of interest in occupational and environmental health:

1) Job-related injuries, illnesses, and fatalities, and
2) accidental spills and uncontrolled waste sites involving hazardous materials.

Historically, public health work in Alaska has emphasized the struggle against infectious diseases. During the 1980s, however, issues of occupational and environmental health have gained increasing prominence. Morbidity and mortality associated with occupational and environmental risks represent a major public health challenge for the future.

Until recently, many Alaska health professionals perceived job safety and health problems almost solely as an enforcement issue falling under the jurisdiction of the Department of Labor (DOL), or otherwise as the concern of management and organized labor. Similarly, many environmental health problems have been relegated to the federal Environmental Protection Agency (EPA) or Alaska's Department of Environmental Conservation (DEC), or seen as primarily "environmentalist" or "conservationist" issues relating more to the preservation of animals and natural resources rather than to the public health of Alaskans.

In part these perceptions have resulted from federal occupational and environmental health legislation which swept across the nation just the decade before, during the 1970s. In the wake of this change, a growing appreciation for the magnitude of occupational and environmental health problems has helped to establish new public health priorities across the nation.

The Seventh Alaska Health Congress, in May 1982, was instrumental in bringing occupational and environmental health to the attention of the Alaskan public health community. A widely recognized public health forum, the 1982 Congress convened around a specific occupational and environmental health concern facing Alaska at the time: the proposed development of a large petrochemical facility in close proximity to Anchorage (2). Subsequent Alaska Health Congresses have featured a range of occupational and environmental health issues reflecting the continuing interest among Alaskan public health professionals.

---

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OVERVIEW OF THE PROBLEM

We reviewed available statistics about job related illnesses, injuries, and fatalaties drawn primarily from *Occupational Injury and Illness Information: Alaska 1986,* a report produced by the Alaska Department of Labor (3). This report, released May 1988, presents the most current occupational health and safety data available for Alaska.

These data, covering both private and public sector workers, come primarily from three sources. First, the Annual Survey of Injuries and Illnesses is a survey designed to produce incidence rates and other information associated with occupational injuries and illnesses by industry. The survey is conducted by the Research and Analysis Section of the Alaska Department of Labor in cooperation with the U.S. Department of Labor's Bureau of Labor Statistics. It draws upon data reported to OSHA by employers. In 1986 a sample of 2,164 representative firms, selected from among approximately 17,000 places of employment, was surveyed by mail or contacted directly.

A second source of occupational injury and illness data reported in the DOL annual publication comes from the Alaska Division of Workers' Compensation. It is known as the Supplementary Data System (SDS). These data supplement the Annual Survey with more detailed information regarding the nature and causes of occupational injuries and illnesses. SDS data are based on workers' compensation claims filed by employees.

A third source of information, the Alaska Fishermen's Fund, was established to provide treatment and care for Alaska licensed commercial fishers who are injured or become ill as a result of commercial fishing in Alaska. Data are summarized from claims received for expenses resulting from work related injuries or illnesses. These data represent an important source of information about occupational injuries and illnesses in Alaska and are reported in the Department of Labor publication. Technically, commercial fishers are not defined as "employees" and as a consequence are not included in either the Annual Survey data or Workers' Compensation data.

OCCUPATIONAL HEALTH AND SAFETY

In 1986, the most recent year for which there is published data, the Alaska Department of Labor reported 13,154 recordable occupational injuries and illnesses for the private sector (4). According to this report the injury rate for all Alaskan private sector workers was 10.2 cases per 100 workers. A recordable occupational injury or illness is defined as any occupational injuries or illnesses which result in:

1) Fatalites, regardless of the time between injury and death, or length of illness;

2) lost workday cases (5). A lost workday case is a case which involves days away from work, or days of restricted activity, or both. The number of lost workdays does not include the day of injury; and,

3) other changes in work status, which meet any one of the criteria:
   (a) result in transfer to another job or termination of employment,
   (b) require medical treatment, other than first aid,
   (c) involve loss of consciousness, or
   (d) result in restriction of work or motion (6).

Manufacturing had the highest rate of occupational injuries compared with other Alaskan industrial sectors in 1986. In manufacturing, there were 28.3 injuries for every 100 workers in 1986, for a total of 3,385 injuries (all rates reported are per 100 workers unless otherwise stated). Within manufacturing two industries contributed most heavily to the high rate of injuries. Seafood processing produced 1,973 cases with an injury rate of approximately 33.3/100 workers. Nearly one of every three seafood workers in Alaska was seriously injured or made ill as a result of working conditions. Alaskan logging camps and contractors recorded and astounding injury rate of 56.6/100 workers. There were a total of 828 cases involving loggers.

The construction industry suffered the second highest incidence rate of occupational injuries, with a total of 1,859 recordable injuries. The average injury rate in this sector of industry was 16.2/100 workers. However, this average obscures some important differences among different types of construction. For example, workers in two major categories, highway and street construction and residential buildings construction, suffered significantly higher injury rates of 20.8/100 workers (264 injuries) and 18.0/100 workers (214 injuries), respectively.

Some specific areas of industry stand out as particularly hazardous, often within larger sectors of industry that evidence a generally lower overall injury rate. Compare, for example, the larger category of retail trade, with an overall rate of 9.2/100 workers (2,552 injuries), with one of its subgroups, food store workers, which had a rate of 18.0/100 (773 injuries). Similarly, the general category of service workers suffered an average injury rate of only 5.4/100. However, workers in hotels and other places of lodging had an injury rate of 13.4/100. High risk groups may be obscured within rates for larger industrial sectors.

In 1986, 9,945 (61%) of the total of 16,208 private and public sector cases were "lost workday" cases (defined above), i.e., more serious injuries. The largest single category among lost workday injuries was sprains and strains at 50% (4,970). Another 12% (1,145) were contusions, crushing, or bruises. An additional 9% (909) were cuts, punctures, or lacerations. Among the most serious were 28 amputations or enucleations, 192 (thermal) burns, 241 dislocations, and 847 fractures, representing another 13% of all injuries.

There were a total of 637 documented lost workday occupational illnesses, representing only 6% of the total time loss cases for 1986. Of these occupational illnesses, 21% (136) were inflamation of the joints, 17% (107) were systemic poisoning, 16% (105) affected the nervous system, 7% (47) were dermatitis and 6% (36) involved mental disorder.

There were a total of 153 occupational illnesses in the manufacturing sector with 82% (125) associated with the food and kindred products industry, almost all in seafood processing. In mining, 25 of 29 (86%) reported lost workday occupational illnesses occurred in the oil and gas extraction branch of the industry.

In 1986, 41 work-related deaths were reported in Alaska. Heart attacks claimed 11 workers. Plane crashes and vehicle accidents each claimed five workers, and four workers drowned. Three workers each were crushed by logs or landslides. The remaining ten died from a variety of causes associated with their working conditions.

It is likely, however, that these data significantly understate the fatalities associated with Alaskan workplaces. A study by Patricia Schnitzer of work-related fatalities in Alaska, 1980-1985, reviewed both workers' compensation records and State of Alaska death certificates. The principal finding concluded that, "Because only workers' compensation data were used to report occupational fatalaties for the state, the Alaska Department of Labor underreported these deaths by 53 percent" (7).

Our third data source, the Alaska Fishermen's Fund, provides information about fishing-related injuries and illnesses. It provides no information about fatalities. However, recent research indicates that from 1981 through 1984, an average of nearly 26 commercial fishers died each year in Alaska, compared to approximately 29 persons in all other industries combined (8). The death rate for commercial fishers in Alaska is 3.2 per 100 worker years, a rate twenty times higher than the average for all other Alaskan industries.

The Alaska Fishermen's Fund received a total of 2,363 claims in 1987. Unfortunately 662 of these were not classified according to nature of injury. Of those classified, 23% (552) claims were for sprains or strains, 20% (463) were for cuts, punctures scrapes, or avulsions, and 8% (178) were for crushing bruises and contusions. Some of the other classifications included 35 cases of carpal tunnel syndrome, 35 cases of respiratory diseases, 167 infectious, and 115 fractures and dislocations, totalling 15% of the classified injuries.

HAZARDOUS MATERIALS IN THE ENVIRONMENT

Incidents involving hazardous materials have become more apparent around Alaska in recent years. Some widely publicized examples include:

- In Moose Pass, 53 people were evacuated from their homes when a railroad tank car containing hazardous materials reacted, leaking toxic vapors. Prevailing winds carried the vapors into residential areas and south into Seward (9). The chemical reaction and subsequent leak actually started while the car was still in Anchorage. Had the car completely erupted while still in Anchorage, many thousands of Anchorage residents would likely have been forced to evacuate.
- Drinking water wells in Peters Creek were contaminated by benzene, a cancer-causing compound, and other chemicals. A leaking underground fuel storage tank is the suspected cause of the contamination (10).
- In the North Slope Village of Wainwright, three residents died after drinking punch spiked with methyl alcohol from a barrel that washed up on the beach (11).
- In Fairbanks, a tank car carrying concentrated hydrochloric acid, a strong corrosive, developed a leak, forcing the evacuation of hundreds of residents (12).
- In the village of Aniak, over 300 gallons of PCB-contaminated oil were found at an old military installation. Some of the drums containing the oil were leaking. An abandoned drum of trichloroethylene, a toxic industrial solvent, was also discovered at the site (13).
- Valley Hospital in Palmer was evacuated after the occurrence of a leak suspected to be ethylene oxide. Used in hospital sterilization equipment, this toxic gas is a known reproductive hazard (14).

As disturbing as these incidents are, they do not present the entire picture. There are thousands of other documented hazardous materials incidents in Alaska. Many more are quite likely never reported. This situation in Alaska reflects the national situation.

A recently released study by the office of Technology Assessment (OTA) notes that most states have serious reporting deficiencies preventing them from responding adequately to hazardous materials incidents. National figures, compiled by the Department of Transportation (DOT), show a decline in the number of hazardous materials incidents on the nation's highways, but these figures are disputed by OTA (15).

A recent investigative newspaper report about hazardous waste in Alaska noted that, "Federal and state regulatory agencies have little data about quantities of hazardous materials transported within Alaska, and lack a comprehensive system for tracking the movement of potentially dangerous substances." Jack Gusmano, a hazardous waste specialist with EPA at the time, said, "We don't generally know what private industry is shipping. There is so much traffic, so much chemical use, you couldn't possibly keep track of it all." Gusmano added that, "There are releases... There is such a vast territory in the state, it could be happening every day and I would not hear about it. Probably in Alaska, the reporting is pretty poor" (16).

In the first study of its type, the Alaska Health Project analyzed hazardous materials incidents in Alaska for one full year, from July 1, 1983 to June 31, 1984. This study adopted the following definition for a hazardous materials incident:

> An incident might be loosely defined as any accident, spill, or event involving a hazardous material which caused or potentially might have caused unnecessary exposure to humans. In operational terms the variety of local, state, and federal agencies that deal with such problems have their own definitions of incidents and hazardous materials. We have typically accepted these definitions without altering them or attempting to modify them for our purposes (17).

Data were compiled from four state and local agency data bases, and from newspaper accounts around the state. A total of 1330 incidents were recorded, with hundreds of these incidents having occurred in the Bush. At least 178 of these incidents involved acute injury or death. The number of cancers and other diseases with long latency periods that developed from exposures associated with these incidents may never be known.

In many cases the quantity of hazardous materials involved in an incident were reported. The total volume in these cases amounts to 590,000 gallons of liquid hazardous materials, plus an additional 460,000 pounds of PCB contaminated materials, a suspected carcinogen.

Of the total 1,330 incidents, 46 involved quantities over 1,000 gallons and totaled about 529,000 gallons of hazardous materials. In other words, about 3% of the incidents involving about 90% of the total materials.

The study divides Alaska into five different regions. Incidents per region were as follows:

Southeast – 250
North – 535
Southcentral East – 498
Southcentral West – 30
Southcentral Aleutians – 17

The U.S. Army Corp of Engineers has identified some 650 sites throughout Alaska that may have been used by the military at one time or another. Of these, attention has been focused on 350 to 400 sites that are suspected of containing hazards such as solvents, glycols, asbestos, old munitions, and oily wastes (18). Many of these hazardous materials are known or suspected carcinogens, and many are explosive or highly reactive. Of importance to rural residents is that many of these sites are located in or near rural communities.

Independent of the Army's efforts, by 1986, the Alaska Department of Environmental Conservation (ADEC) and the U.S. Environmental Protection Agency (USEPA) had compiled a joint list of 152 potential uncontrolled hazardous waste sites in Alaska. About two dozen of these sites, some of which include the Nome City Dump, Nome Barrel Dump, the city dump at Fort Yukon, waste ponds in Bethel, and Standard Steel and Husky battery sites in Anchorage, are still under investigation (19).

A 1985 study of potential hazardous waste sites in Alaska by Tetra Tech noted that the stockpiling of explosive and hazardous materials is a "regular practice in remote locations." In addition, Tetra Tech reported, "... the accumulation of large numbers of drums that may then be used to store wastes. When a remote (industrial) site is abandoned, these products are often left behind. Major oil companies operating on the North Slope have conducted regular clean–ups of such inventories, and have deposited waste materials into nearby landfills. These sites do not always meet RCRA (Resource Conservation and Recovery Act, Environmental Protection Agency) standards for hazardous waste disposal, and must be closely monitored" (20).

In 1983, TXT Limited completed a survey of hazardous materials shipped through Alaskan seaports (21). The report identified types and volumes of hazardous materials shipped through various ports in Alaska, documenting that thousands of gallons of hazardous materials are regularly transported to rural areas in the state. Some of these materials such as chlorine, ammonia, and hydrochloric acid may require isolation or evacuation of the public if accidentally released. Tens of thousands of gallons of explosive gases, and thousands of pounds of explosives and munitions are shipped through bush Alaskan ports annually.

During a one-year period ending October 31, 1985, the Alaska Railroad shipped 772,473 tons of hazardous materials in 14,383 rail cars (22). Some of the materials shipped included:

- Military related ammunition and explosives—578 tons
- Compressed or liquid gas – 3,860 tons
- Sulphuric and other acids – 4,559 tons
- Non–military explosives – 533 tons
- Jet fuel, kerosene, and blended gasoline – 343,765 tons
- Miscellaneous chemicals – 1,468 tons
- Butane and LPG propane gas – 11,573 tons

DISCUSSION AND RECOMMENDATIONS

Data reported above provide the most comprehensive overview of selected occupational and environmental health concerns currently available. However, it must be recognized that no adequate reporting system exists, and that these data should be interpreted with some caution. For instance, in 1985 the Bureau of Labor Statistics contracted with the National Academy of Sciences to review the Bureau's statistical programs for work-related injuries and illnesses (23). Some of their major findings include:

**Employers may be reporting minor cases that do not require reporting. As a result, some incident rates derived from the Annual Survey may be overstated. The problem may be the lack of understanding among some employers about what constitutes a reportable incident.

**On the other hand incident rates may be underreported by some employers. In 1981 the Reagan administration changed the criteria for implementing OSHA compliance inspections. Originally they were based on industry wide incident rates. After 1981 individual enterprises with time loss case rates below the industry wide average receive only a "paper inspection" rather than an on site inspection. This policy creates an incentive to underreport serious occupational injuries and illnesses. A number of such cases have been found and prosecuted in recent years.

**Fatality data may be underreported since the Alaska Department of Labor has until now only used workers' compensation data to determine work related fatalities. The workers' compensation system is inadequate to pick up many fatalities resulting from chronic work related diseases.

**Similarly, the Annual Survey is better at revealing acute illnesses clearly related to work, e.g., contact dermatitis or systemic poisoning. Diseases with long latency periods are probably underreported. This is particularly so since fishing is a seasonal type of work with a highly mobile workforce.

Data on both specific hazardous materials releases and the resulting immediate and/or long term impact on public health in Alaska is largely anecdotal, given the lack of the systematic documentation characteristic of health and safety data. Much of the general information is hardly more than guesswork. Limited resources among state agencies, a chaotic mix of public agencies with no standard reporting criteria or systems, and inconsistent and underutilized federal/state reporting systems contribute to inconsistent data.

More than one out of every ten Alaskan workers in the private sector is seriously injured or made ill as a result of their work conditions each year. In some industries the risk is much higher. Seafood processing, for example, is a high growth industry in contemporary Alaska. Each year in this industry approximately one of every three workers is seriously injured or diseased as a result of conditions at work. Clearly this is a public health issue of the first order, deserving a larger proportion of our time, analysis, service.

The public health threat of accidental spills and uncontrolled waste sites involving hazardous materials is vastly more complicated to assess. There is evidence, for example, that the water supplies in a number of Alaskan communities are seriously contaminated by known or suspected carcinogens. The Moose Pass incident demonstrated the potential disaster for an entire community when toxic chemicals are accidentally released to the air. The hundreds of known and suspected hazardous waste sites across the entire state represent a public health threat of unknown proportions. In addition many hundreds of thousands of pounds of hazardous materials are transported across Alaska every year presenting potential problems to the public health. Given all of this, state and federal officials readily admit to their inability to keep track of transportation, incidents, or the public health impact of hazardous materials in Alaska.

Without an adequate database, we can only speculate about the overall threat posed by hazardous materials. It is time to establish a systematic approach to reporting and compiling the evidence on existing hazardous waste sites and releases of these materials into the environment. A better database is required, one at least as effective as that currently in place for occupational health and safety. In addition, these problems deserve far more research regarding the potential threat to the public health in Alaska. Public health professionals must have better information on which to base a comprehensive plan of action to protect the public from hazardous materials and hazardous waste in Alaska.

Recent passage of Title III of the Superfund Amendments and Reauthorization Act of 1986 may provide the state with a foundation upon which to build a better data collection and emergency response system relating to hazardous materials. Title III requires that the state develop coordinated hazardous materials emergency plans. Furthermore the legislation requires that databases be developed to keep track of hazardous materials in Alaska and document toxic chemical releases.

We should note that Title III includes provisions for public access to this information, reflecting increasing awareness and concern nationally. This can only lead to a more meaningful debate about hazardous materials and a more responsive risk assessment process. With an adequate database the potential victims of toxic releases can have a role to play in the decision making process regarding the hazardous materials in Alaska.

In conclusion, however, as we reflect upon the efforts of the last five years to protect the public health of Alaska, it is clear that we have yet to implement a coordinated agenda with specific plans to address occupational and environmental health needs in Alaska.

ACKNOWLEDGMENTS

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24. Personal communication with Mark Catlin, July 15, 1988.

A Survey of Indoor Air Quality in Twenty-Nine Anchorage Residences

by Richard L. Myers, Ph.D.[1]

ABSTRACT

During the winter of 1987–88, 29 Anchorage residents who contacted various agencies with indoor air pollution inquiries had their homes inspected and monitored for several indoor air pollutants. A survey form was completed during an interview with occupants of each residence visited. Data were collected on house characteristics and behavior of occupants. The mean nitrogen dioxide (NO_2) concentration for residences with measurable amounts of this gas was .024 ppm, while the mean formaldehyde (HCHO) concentration with measurable levels was .037 ppm. Residences with gas cooking ranges had mean NO_2 concentrations twice as high as those with electric ranges. Several residences had NO_2 concentrations exceeding recommended standards.

INTRODUCTION

Indoor air pollution has emerged as a major public health issue during the past decade(1). Approximately 90% of our daily activities occur indoors(2). Because of Alaska's climate, the proportion of time spent inside is probably even higher in Alaska. This places added importance on a healthful indoor environment in Alaskan homes. Though geography dictates greater emphasis on indoor air quality in our state, indoor air studies in Alaska have been episodic and primarily in response to complaints voiced by public building occupants. Examples of the latter include the state department of labor building in Anchorage during 1985 and Wasilla's Finger Lake School episode during 1987–88.

As more individuals become aware that indoor pollution in their homes can lead to health problems, an increasing number seek assistance from governmental agencies. Unfortunately, these agencies often do not possess the resources to respond to residents' requests for assistance. Because the lack of resources limits the scope of government's response to indoor air pollution complaints, there is a dearth of information concerning indoor air quality in Alaskan residences.

The primary goal of this study was to assess indoor air quality in Anchorage residences. During the winter 1987–88, I responded to approximately 50 referrals from various groups that typically receive inquiries from home owners or renters about indoor air quality. I collected data on the nature of the problem, characteristics on the home and its occupants, and concentration of nitrogen dioxide (NO_2). formaldehyde (HCHO), and in several cases carbon monoxide (CO).

METHODS

From November 1987 through March 1988, several agencies referred individuals with indoor air pollution concerns to me. Referrals came primarily from the American Lung Association of Alaska, Municipality of Anchorage, and Alaska Health Project. Upon referral, I contacted the individuals and made a preliminary assessment of the situation. Some individuals had concerns which were not within the scope of the study, and I suggested that they contact other sources for help. Specifically, those who requested information about radon testing were told to contact the Alaska Department of Health and Social Services. This agency has been conducting a statewide radon survey for the past several years. Individuals having problems with natural gas combustion appliances were directed to Enstar natural gas company of Anchorage.

From the referrals, I made visitations to 29 residences. During the visit, I conducted an interview with home occupants and completed a survey. The survey was used to determine the nature of the problem, to collect information on house characteristics, and to collect information on occupants and their habits. Data collected for each residence included type of construction, age, size, type and location of heating system, cooking fuel, fireplace/woodstove use, exposure, and ventilation. Data on occupants included number of adults, children, and pets, smoking habits, and daily activities in the home. After the interview, I placed two NO_2 monitors in each residence. One was placed in an area with the highest occupant activity and the other placed in an area with the highest expected concentration, for example, in a kitchen over a gas stove. When the areas of highest activity and concentration were thought to be the same, the second monitor was placed in a different liveable portion of the residence, for example, upstairs if the first monitor was placed downstairs. I also placed a HCHO monitor in most of the residences.

Air Quality Research (AQR) of Berkeley, California provided the monitors and performed the analyses. AQR manufactures a variety of indoor air sampling monitors specifically suited for residential use. The NO_2

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monitors are identical to those originally designed by Palmes and have been used extensively in indoor air quality studies(3). AQR HCHO monitors are used by a number of organizations conducting indoor air monitoring such as Oak Ridge National Laboratories and Bonneville Power Authority. The State of Massachusetts selected AQR monitors to perform required testing under their new urea-formaldehyde, foam insulation statute. Table 1 summarizes characteristics of the NO_2 and HCHO monitors.

Table 1. Monitor Characteristics

	NO_2	HCHO
sampling time	5–7 days	5–7 days
minimum detection limit	.008 ppm	.010 ppm
maximum detection limit	.5 ppm	1 ppm
accuracy	± 25%	± 30%

In several residences, I measured instantaneous CO concentrations with an Ecolyzer 2000 series CO monitor. I employed a sling psychrometer and used the wet-dry bulb method to determine relative humidities.

RESULTS

Twenty-one of the 29 residences in the sample were single family detached houses, six were condominiums, and two were duplexes. Residences ranged in size from 650 square feet to 5,000 square feet with a mean size of 1,900 square feet. Twenty-five of the homes had either natural gas boilers or furnaces for heating. Two were heated electrically, one used a combination of wood and propane, and one used an electric space heater and an oven for heat. Sixteen homes had electric ranges, and 13 had gas ranges. The mean age of the 29 residences was just under 16 years and ranged from 0 to 60 years. Occupancy of the homes ranged from 0 (vacant) to one home with three adults and five children; mean occupancy was 3.1 individuals. Nineteen of the residences contained pets. Two residences had occupants who smoked at least one pack of cigarettes per day in the home (Table 2).

Two NO_2 in each residence gave a total of 58 samples. Twenty of these samples registered below the monitor's minimum detection limit of .008 ppm. The mean concentration for the remaining 38 monitors was .023 ppm with a standard deviation of .026 ppm. The mean NO_2 concentration detected by 22 monitors in residences with gas ranges was .030 ppm. NO_2 concentration detected by 16 monitors in residences with electric ranges

Table 2. Characteristics of 29 Residences

characteristic	mean or number	s.d.	range
type:			
single family detached	21		
condominium/apartment	6		
duplex	2		
size	1920 sq. ft.	930	650–5,000
age	16 years	15	0–50
heating:			
natural gas boilers	12		
natural gas furnace	13		
electric baseboard	2		
wood/propane	1		
electric space	1		
cooking:			
electric range	16		
gas range	13		
occupancy	3.1 individuals	1.4	0–8
pets:			
0	8		
1	13		
2	7		
3	1		
relative humidity	48%	10	32–68
smoking:			
0 cigarettes/day	24		
1–20	3		
21–40	2		

was .015 ppm. The highest NO_2 concentration measured was .147 ppm. Eight residences had both NO_2 monitors register below detectable limits, and seven of these were in residences with electric ranges. Table 3 displays monitor results for each individual residence and Table 4 summarizes results from all the monitors. Figure 1 compares NO_2 results in residences with gas and electric ranges.

I placed a single HCHO monitor in 24 residences and two monitors in three homes. Two homes were not tested for HCHO. Only one residence had a HCHO concentration below the detectable limit of .010 ppm. The mean concentration for the remaining 29 monitors was .037 ppm with a standard deviation of .024 ppm. HCHO concentrations ranged from .013 to .126 ppm.

DISCUSSION

Individuals referred to me reported a variety of concerns and symptoms that they perceived were related to poor indoor air quality. As mentioned previously, some had specific concerns about radon and combustion equipment. I suggested to these individuals that they contact agencies or groups which dealt with their specific problem. The majority of individuals referred to me could be classified into two groups. One group consisted of individuals not experiencing health problems who had a general interest in indoor air quality. Individuals experiencing health problems and seeking relief comprised the second group. Most in this second group reported common symptoms associated with indoor air pollution: excessive coughing, sneezing, watery eyes, headaches, lethargy, and congestion. Several individuals strongly felt their health problems were directly associated with their residence and reported that their health improved when they vacated their primary residence for an extended period of several days or more.

The inspections and limited monitoring revealed several homes where poor indoor air quality might be contributing to the health problems reported by the occupants. In one home, NO_2 concentrations were .147 ppm in the living room and .078 ppm in the basement; the National Ambient Air Quality Standard (NNAAQS)

Table 3. NO_2 and HCHO Concentrations in 29 Anchorage Residences

number	NO_2 (main living area)	NO_2	HCHO (main living area)	HCHO
1	.014	nd[1]	nd	
2	nd	nd	.016	
3	nd	nd	.018	
4	.010	.012		
5	.009	.009	.019	
6	nd	nd	.055	
7	.008	.008	.038	
8	.019	.040	.030	.043
9	.013	.010		
10	nd	nd	.025	
11	nd	nd	.088	
12	.013	.010	.020	
13	.023	.023	.038	
14	nd	nd	.013	
15	nd	nd	.033	
16	nd	.012	.016	
17	.022	.024	.031	
18	nd	.011	.027	
19	.032	.025	.016	
20	.008	nd	.030	
21	.028	.014	.053	.041
22	.008	.012	.042	
23	.019	.012	.042	.126
24	.022	.013	.043	
25	.025	.015	.061	
26	.029	.026	.028	
27	.020	.065	.027	
28	.147	.078	.054	
29	nd	nd	.016	

all concentrations expressed in ppm **[1]nd=not detectable** **numbers 1–16 are residences with electric ranges**

Table 4. Summary of Monitoring Results

	n	mean	standard deviation	95% confidence limits	range
NO_2	38	.023	.026	.015-.032	.008-.147
HCHO	29	.037	.024	.029-.045	.013-.126

all concentrations expressed in ppm

for NO_2 is .05 ppm and is often used as a guideline for healthy indoor air. The occupants in this home were an elderly couple who experienced coughing and other respiratory illness. They were using their oven, which was not vented outdoors, as a primary heat source. Their condition improved when they stopped using the oven for heat and opened a window slightly to increase ventilation.

The HCHO concentrations measured in this study corresponded to those found in other studies on residences that do not contain urea formaldehyde foam insulation (4,5). Two homes had HCHO concentrations which approached 0.1 ppm. Massachusetts, Canada, Sweden, Netherlands, and West Germany have established an indoor HCHO standard of 0.1 ppm. A concentration above 0.1 ppm (.126 ppm) was actually measured in one residence, but this was in a room used for storage. The HCHO concentration in the main living area of this house was .042 ppm. The owner reported experiencing chronic flu-like symptoms since moving into the house in 1985. She also mentioned that several visitors experienced sickness when staying in the house. This residence is 30 years old, and a HCHO source in the house could not be located.

Another home owner reported moving into an 11-year old house in July (1987) and immediately experience headaches, stomach aches, breathing problems, and a cough. This home had new carpet, a material known to emit HCHO, installed just before the owner took occupancy. I obtained a HCHO concentration of .088 ppm in the living room of this house. The individual shared the results of monitoring with her physicians who she was seeing about her health problems.

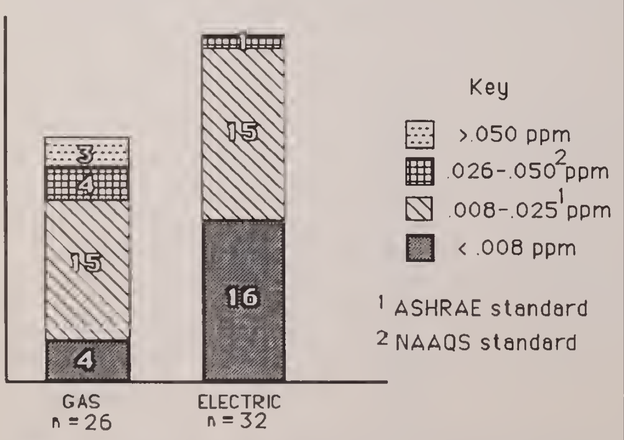

Figure 1. NO_2 concentrations in residences with gas and electric ranges.

Complaints reported in the literature associated with HCHO exposure include eye, nose, and throat irritation, headaches, and skin rash (6). These symptoms are experienced by a small percentage (10%) of individuals when exposed to concentrations at 0.1 ppm and below (7). While HCHO can not be totally ruled out as responsible for some of the health complaints reported by individuals in this study, most residences had HCHO concentrations that are unlikely to account for their health complaints.

A carbon monoxide (CO) problem was found in one residence. Occupants reported odors in their apartment when an adjoining unit's fireplace was in use. Instantaneous CO levels measured over a 30-minute period ranged from to 10 to 14 ppm; NAAQS for CO is 9 ppm averaged over 8 hours. After opening a window in the apartment for 20 minutes, the CO concentration decreased to 2 to 3 ppm. Occupants of this unit consisted of a mother and newborn twins. Because of my inspection, they moved to a different complex a week after I visited their apartment. Results of the inspection were shared with the landlord, and it was noted that a fireplace backdrafting problem might exist.

Several researchers found that the type of cooking appliance is a major factor controlling residential NO_2 concentrations (3,7). Residences with gas ranges generally have NO_2 concentrations several times higher than residences with electric ranges. My results tend to support this. Sixteen of the 20 monitors which registered below the minimum detection limit of .008 ppm were in electric range residences, even though were 16 electric range homes compared to 13 gas homes. Five houses with gas ranges had at least one measured NO_2 concentration above the recommended American Society of Heating and Refrigeration Engineers (ASHRAE) standard of .025 ppm. Only one house with an electric range had a NO_2 concentration above .025 ppm.

The mean NO_2 concentration in residences with gas ranges was .030 ppm. The mean for residences with electric ranges was .015 ppm. A one tail t-test supported the hypothesis that NO_2 concentrations were higher in residences with gas ranges (p=.031). Even when the residence using a gas range in an unconventional manner as a heat source is eliminated, the difference in NO_2 concentrations between gas and electric range residences is still statisically significant (p=.030).

CONCLUSION

While many residents believe that indoor air quality is affecting their health, it is extremely difficult to identify problems based on limited inspections and monitoring. The myriad of indoor pollutants, home characteristics, and occupants' habits are just a few factors which make each case unique. It would help if residents educated themselves on the basics of indoor air quality. The State of Alaska is taking several steps to help with this education process. A recently completed series of eight fact sheets on indoor air pollution with emphasis on Alaska and the American Lung Association of Alaska. A 20-minute video entitled *The Air We Breathe* is also available through the State Department of Community and Regional Affairs.

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Pedestrian Fatalities in Alaska, 1980–1984

by John P. Middaugh, M.D.[1]

ABSTRACT

During 1980–1984, 94 pedestrians (15.6% of 601 total traffic-related fatalities) were killed and 1,293 were injured in Alaska. The pedestrian fatality rate for Alaskan Natives (10.95 per 100,000 population per year) was four times the rate for non-Natives (2.84 per 100,000 population per year), and in Anchorage was 13 times higher (37.98 per 100,000 per year compared to 2.89 per 100,000 per year, respectively). Alcohol use by adult pedestrians and drivers was strongly associated with pedestrian fatal events and was especially associated with Alaskan Native pedestrian fatalities (O.R. 8, 95% CI 1.95–32.78). This previously unrecognized epidemic needs to be controlled by attacking associated risk-factors and by modifying the environment to separate pedestrians from motor vehicles.

INTRODUCTION

Intentional and unintentional injuries are the leading causes of death in Alaska. Motor vehicle–related fatalities are the leading cause of injury deaths, accounting for 21.6% of all fatalities caused by injuries in Alaska. In 1983, the Epidemiology Office, Division of Public Health, began to investigate intentional and unintentional injuries in Alaska. With the support of the Section of Communications and Information, Division of Plans and Programs, Department of Transportation and Public Facilities, the Epidemiology Office initiated this study of pedestrian fatalities in Alaska.

Injuries are not accidents. Accidents, by definition, are random and unpredictable events—therefore, they are unpreventable events. Injuries are not randomly distributed and often are predictable. If risk factors associated with injuries can be identified, specific control measures can be implemented with great success. Most injuries can be prevented (1).

METHODS

All Alaskan death certificates for 1980–1984 (N= 9,361) were manually reviewed. All death certificates listing the cause of death as being related to a pedestrian fatality were identified, and data were extracted from the death certificate. Data tapes from the Department of Transportation and Public Facilities (DOT&PF) were obtained and analyzed to identify all motor vehicle collisions involving pedestrian fatalities. Data were obtained to characterize the drivers, vehicles, and environmental conditions involved in the fatal pedestrian event. Alaska law requires that the Department of Public Safety (DPS) and DOT&PF receive all police accident reports for all incidents in which a person is injured or in which property damage exceeds $500. Reports from Alaska State Troopers and all local police were forwarded to DOT&PF and DPS for editing, coding, and computer entry. All pedestrian fatalities identified from death certificates were matched, when possible, with the corresponding DPS/DOT data file. Death certificates were obtained for every pedestrian fatality identified from DPS/DOT data tapes. Records of the three state medical examiners were reviewed to obtain blood alcohol determinations.

Alcohol use by pedestrian victims was determined by the presence (blood alcohol concentration > 0) of alcohol in blood samples obtained at autopsy (N = 37), by a police breathalyzer test at the scene (N = 1), and by the investigating officer's opinion (N = 1). Alcohol use by drivers was determined according to police accident reports when the investigating police officer's opinion was that alcohol use by the driver was the contributing cause (N = 7), if a citation was issued for alcohol intoxication (N = 10), or if an alcohol level was recorded (N = 15).

Statistical analyses were done using the Fisher's Exact Test and the Mantel–Haenszel Test, using the Statistical Analysis System (SAS).

RESULTS

During 1980–1984, there were 94 pedestrian fatalities and 1,293 pedestrian injuries (from DOT records only) in Alaska. For comparison, in 1980, there were 9,600 pedestrian fatalities and 120,000 pedestrians injured in the United States (2). The ratio of pedestrian fatalities to injuries in Alaska (7.3%) was similar to the United States (8.0%). During 1980–1984, the 94 pedestrian fatalities constituted 15.6% of the 601 total traffic-related fatalities in Alaska.

During 1980–1984, the average annual rate of motor vehicle–traffic (E810–E819) fatalities was 18.3 per 100,000 for Natives and 17.3 per 100,000 for non-Natives. For comparison, the motor vehicle–traffic fatality rate for the total U.S. in 1977–1979 was 22.37 per 100,000 and for Native Americans was 50.88 per 100,000 (2).

The fatality rate for Alaskan Natives (10.95 per 100,000 per year) was 3.8 times the pedestrian fatality rate for Alaska non-Natives (2.84 per 100,000 per year).

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Table 1. Pedestrian Fatalities in Alaska, 1980–84 Fatality rates per 100,000 population per year

Age Group	Native	Non-Native	Alaska Total	USA* 1977–79
0–4	4.65	2.57	2.95	0.22 3.88
5–9	8.72	2.45	3.54	4.42
10–14	2.72	4.32	4.03	2.23
15–19	9.40	1.91	3.51	3.44
20–24	16.22	3.12	5.24	3.33
25–29	9.55	2.70	3.44	2.72
30–34	11.22	2.92	3.75	2.33
35–39	15.24	1.74	3.13	2.56
40–44	12.61	0.83	2.20	
45–49	21.41	1.16	3.98	3.34
50–54	32.28	4.06	8.15	
55–59	29.93	1.67	5.73	3.86 5.31
60+	4.24	9.07	8.05	9.73 9.72
Totals	10.95	2.84	4.06	3.58

* Baker SP, O'Neill B, and Karpf RS. The Injury Fact Book, © 1984, p285.

Except for children in the 10–14 year age group and adults older than 60 years, Native pedestrian fatality rates exceeded non-Native rates for all age groups (Table 1). Although non-Native, age-group specific pedestrian fatality rates in Alaska were similar to national rates, Native pedestrian fatality rates exceeded both non-Native rates in Alaska and national rates in all age groups (Table 1).

The number of pedestrian fatalities increased each year from 1980 to 1983 but decreased slightly in 1984 (11, 15, 18, 28, 22, respectively). In every year, more male than female pedestrians were killed. Caucasians accounted for 52 (55%), Natives 39 (41%), Koreans 2 (2%), and Blacks 1 (1%) of pedestrian fatalities. Pedestrians of all ages died. The largest number of fatalities occurred in the 20–34 year age group with a surprisingly large number of fatalities in individuals 60 years and older (Figure 1). Of the 94 fatalities, 45 (48%) occurred in Anchorage.

Little variation occurred in the distribution of pedestrian fatalities by month or day of the week. Most pedestrian fatalities occurred between 4:00 P.M. – 8.00 P.M. and in the one hour before midnight. The fewest number occurred between 8:00 A.M. – noon. Of the 94 fatalities, 54 occurred at night; 30 when it was dark, and 24 when it was dark but near street lights. Only 14 (15%) of the 94 fatalities occurred when there was fog, snow, or rain; and 44 (47%) fatal events occurred under dry road conditions. The road was straight and level or straight with a grade in 73 (78%) of the 94 fatalities.

In none of the 94 fatalities was the vehicle making a right turn after stopping at an intersection for a red light. In 72 (77%) cases the pedestrian was killed on the roadway. There was no traffic control signal in 77 (82%) of the 94 events.

In each year, male drivers of the vehicles involved in the fatal pedestrian collision were much more numerous than female drivers. Male drivers in the 20–24 year age group most frequently were involved in fatal events.

Information on alcohol use by pedestrian victims was exceptionally accurate. Of 72 pedestrian fatalities 15 years and older, autopsies were performed on 67 (93%) and toxicology results were available on 55 (76%)—37 had alcohol in their blood. Natives and non-Natives 15 years and older had similar autopsy (91% and 95%) and toxicology rates (84% and 70%, p = 0.125, Fishers Exact test) (Table 2). Two additional fatalities were included as alcohol-related based on breathalyzer results and the investigating officer's opinion, respectively. Of 22 fatalities less than 15 years of age, 20 (91%) were autopsied but toxicology tests were done only on 5 (23%). Of the 17 children less than 15 years of age who did not have toxicology tests done, 11 were less than 15 years of age who did not have toxicology tests done, 11 were less than 10 years old, and two of the remaining six were brothers killed while delivering newspapers early in the morning. All five children tested had blood alcohol levels of zero.

Data on alcohol use was found primarily in autopsy reports of the medical examiners (N = 37). Police accident reports detected only 12 (35%) of the 34 alcohol-related pedestrian fatalities from the 86 fatalities in DOT records. Medical examiner records contained toxicology reports documenting positive blood alcohol levels in 32 (94%) of the 34 cases known to DOT. We discovered eight additional pedestrian fatalities not recorded in DOT records by reviewing death certificates—medical examiner records contained positive alcohol levels in three of these cases. There were several instances where toxicology tests were not done because of the length of time from the crash to autopsy.

Of striking importance was the association of alcohol use by both pedestrians and drivers in fatal events. Of pedestrian victims who had alcohol detected at autopsy, only 5 of 38 had levels less than 0.1 g/dl, and the mean and median levels were almost twice that level in all groups (Table 3). Among pedestrians, 13 (48%) of 27 females and 26 (50%) of 52 males for which data were available were using alcohol at the time of their death, based on presence of detectable blood alcohol at autopsy. Among those using alcohol, 22 (56%) of 39 were Alaskan Natives.

Figure 1. Fatal Pedestrian Vehicle Crashes in Alaska 1980–1984
by age and alcohol use by the pedestrian
N = 94   N (alcohol) = 39   N (no alcohol) = 39   N (unknown) = 16

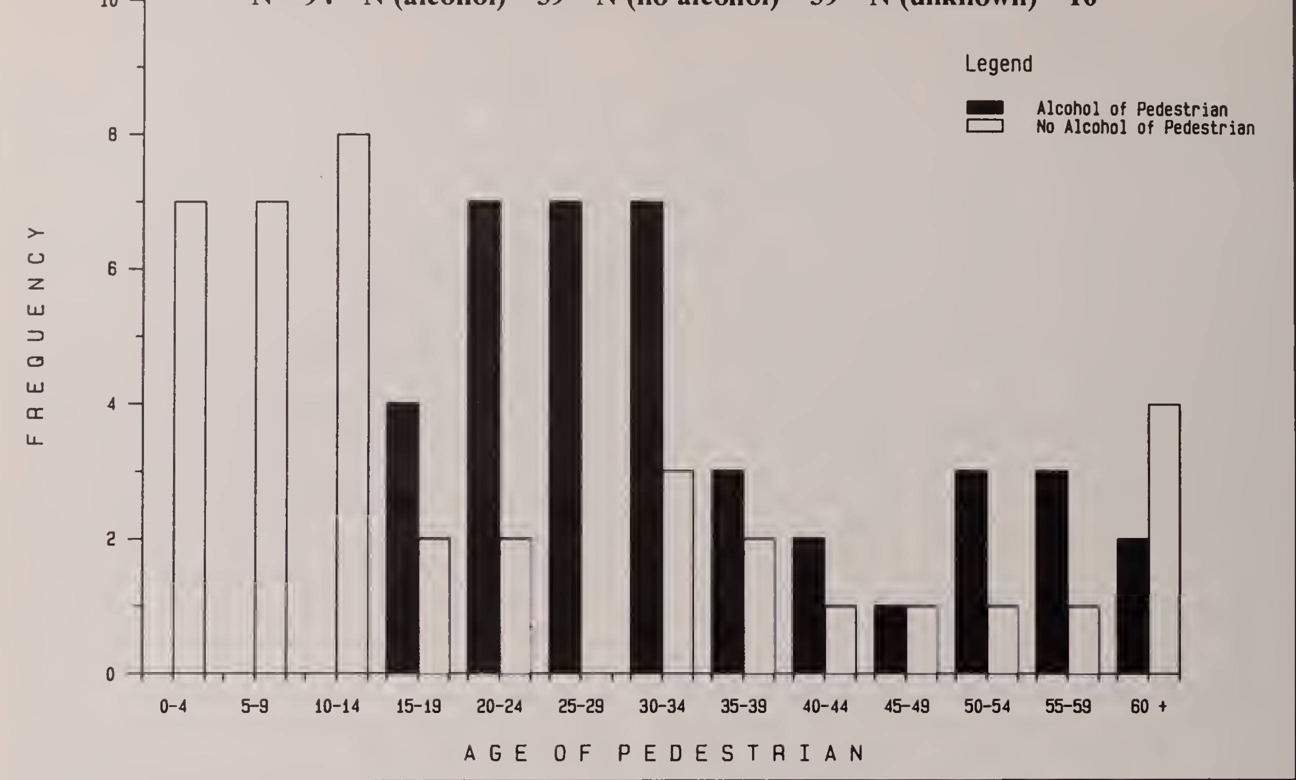

Table 2. Number of Autopsies and Toxicology Tests by Age Group and Race

Age Group		Native Toxicology	Native Autopsy	Native Total	Non-Native Toxicology	Non-Native Autopsy	Non-Native Total	Total Toxicology	Total Autopsy	Total
Age (years)	≥ 15	27	29	32	28	38	40	55	67	72
	< 15	2	4	6	3	16	16	5	20	22
Total		29	33	38	31	54	56	60	87	94

Table 3. Blood Alcohol Levels Among Adult Pedestrian Fatalities with a Positive Test Result

	Number of Positive Tests	Mean (g/dL)	Median (g/dL)	Range (g/dL)	Number of Positive Tests < 0.1 g/dL
Native Male	12	0.206	0.207	0.019–0.468	2
Non-Native Male	13	0.233	0.184	0.010–0.533	2
Native Female	12	0.212	0.218	0.030–0.390	1
Non-Native Female	1	0.200	0.200	0.200	0

Not unexpectedly, alcohol was not found in pedestrians killed who were less than 15 years of age. Among individuals for which information was available, a large proportion of adults, including 24 (77%) of 31 pedestrians 20–39 years old, were using alcohol at the time of their death (Figure 1). For pedestrians 15 years and older, Alaskan Native pedestrian victims were more likely to be using alcohol than non–Natives (p=0.0124, Fisher's Exact Test; O.R. 8.0, 95% C.I. 2.41–29.89) (Table 4). Adult Native pedestrian victims in Anchorage also were using alcohol more frequently than non–Natives (85% vs. 53%) but the difference was not statistically significant (p=0.086, Fisher's Exact Test; O.R. 4.8, 95% C.I. 0.82–28.27).

Table 4. Fatal Pedestrian Vehicle Collisions in Alaska, 1980–84 Alcohol Involvement by Race for Pedestrians 15 years and older.

Race	Alcohol Present	Alcohol Absent	Total
Native	24	3	27
Non–Native	15	14	29
Total	39	17	56

Fisher's Exact Test, P = 0.0026

Mantel–Haenszel, OR=8.0; 95% CI 2.14–29.89

There were 96 drivers involved in 92 crashes that accounted for the 94 pedestrian fatalities. Of 78 drivers where data were available, 19 (30%) of 64 males and 4 (29%) of 14 females were known to have been using alcohol at the time of the crash. Alcohol use by the driver was most frequent in drivers in the 16–19 and 20–24 year age groups (Figure 2).

We were able to match data about the driver and pedestrian involved in each event. Alcohol was known or strongly suspected to be involved in 23 drivers and 38 pedestrians involved in 92 fatal pedestrian crashes. In 13 events, both the driver and pedestrian were using alcohol (Table 5). Pedestrians less than 15 years of age, none of whom were using alcohol, were not more likely to be struck by a driver using alcohol than were pedestrians older than 15 years of age.

Because 48% of all pedestrian fatalities occurred in Anchorage, we analyzed Anchorage data separately. Characteristics of the drivers and pedestrians were similar to those presented for the State of Alaska. However, the rate of pedestrian fatalities in Anchorage for Alaska Natives (37.98 per 100,000 population per year) was 13 times higher than the fatality rate for non–Natives (2.89 per 100,000 per year). The locations of fatal pedestrian events and those known to be associated with alcohol use by the pedestrian in Anchorage are shown in Figure

Figure 2. Fatal Pedestrian Vehicle Crashes in Alaska 1980–1984 by age and alcohol use by the driver
N = 96 N (alcohol) = 23 N (no alcohol) = 59 N (unknown) = 14

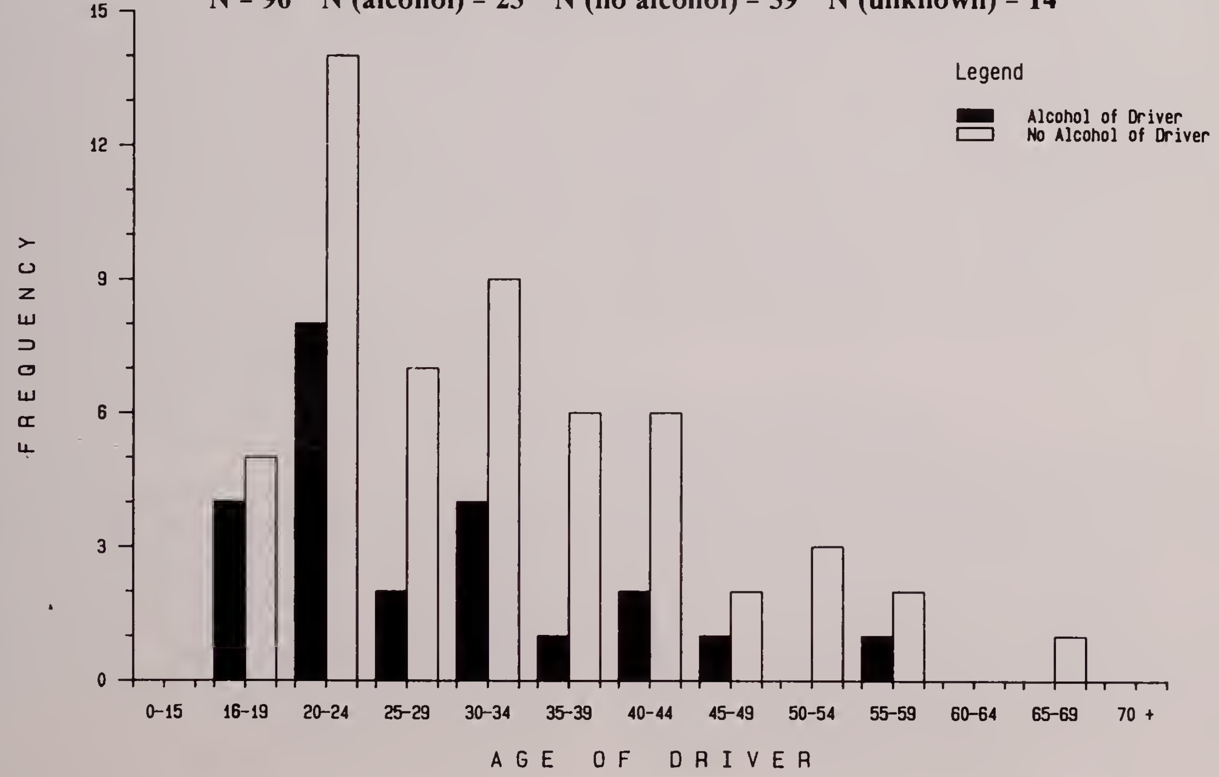

Table 5. Fatal Pedestrian Vehicle Crashes* in Alaska, 1980-84 by alcohol involvement of the driver and pedestrian

		Driver			
		Alcohol	No Alcohol	Unknown	Total
Pedestrian	Alcohol	13	19	6	38
	No Alcohol	8	25	5	38
	Unknown	2	11	3	16
	Total	23	55	14	92

*Note: Data based on crashes. In one crash, there were two pedestrians killed - both had positive blood alcohol tests at autopsy.

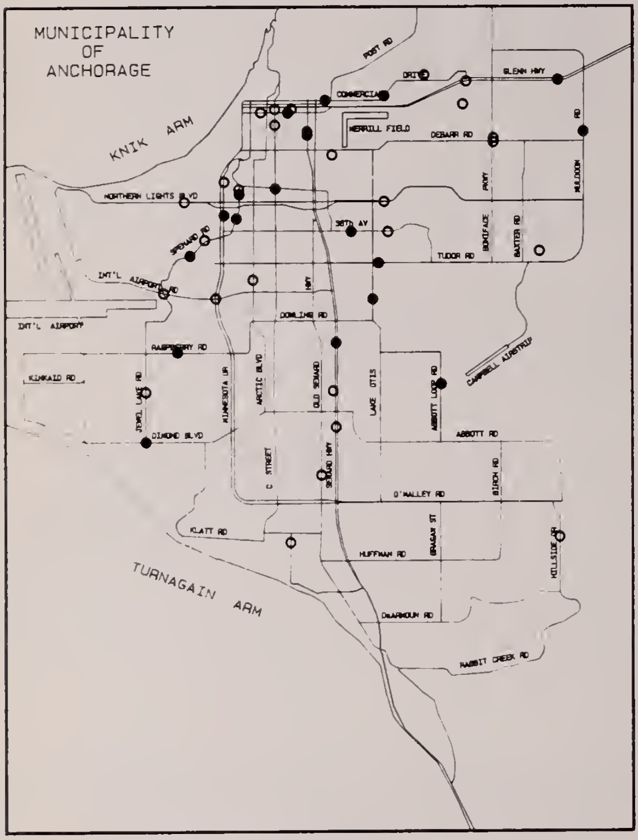

Figure 3. Fatal Pedestrian Vehicle Crashes in Alaska 1980-1984

● Pedestrian Using Alcohol

○ Pedestrian Not using Alcohol

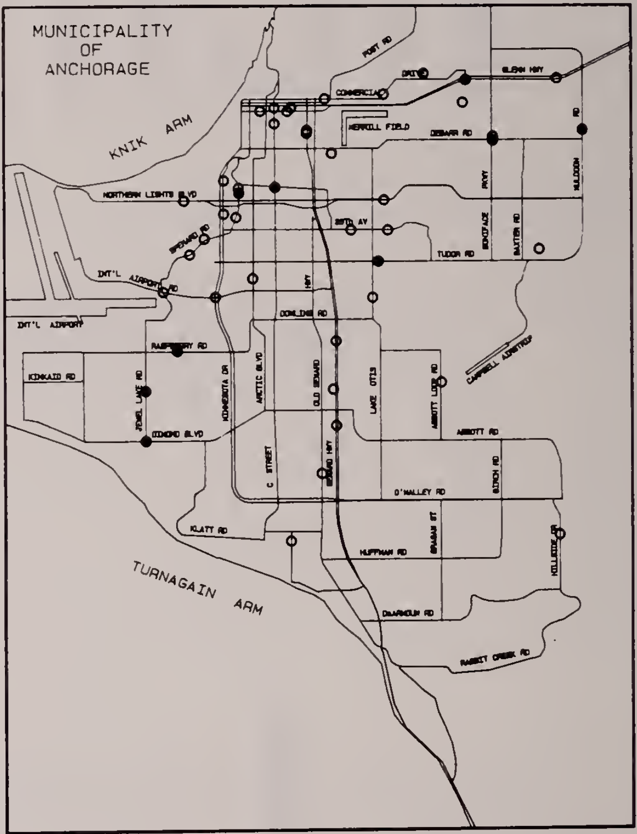

Figure 4. Fatal Pedestrian Vehicle Crashes in Alaska 1980-1984

● Driver Using Alcohol

○ Driver Not using Alcohol

3, and those associated with alcohol use by the driver in Figure 4.

DISCUSSION

The overall Alaska pedestrian fatality rate was only slightly higher than the pedestrian fatality rate reported for the U.S. population. While the total motor vehicle–traffic fatality rates for Alaska Natives, non–Natives, and the total U.S. were similar, the Alaska Native rate was almost three times lower than the U.S. reported rate for all Native Americans. However, the pedestrian fatality rate for Alaska Natives was almost four times the rate for non–Natives, and in Anchorage, the Alaska Native pedestrian fatality rate was 13 times higher than the rate for non–Natives. This largely unrecognized epidemic of pedestrian fatalities among Alaska Natives cries for attention and effective action.

Alcohol use by both pedestrians and drivers was a highly associated risk factor in fatal pedestrian events. In Alaska, young adult pedestrian victims are overrepresented compared to national data, and 77% were using alcohol at the time of their death. Alcohol use is a major contributer to the increased pedestrian fatality rate among Alaska Natives.

In the past 10 years, there have been increases both in population and in motorization in Alaska. Risk of pedestrian fatality can be defined in straight epidemiologic parameters of age-, sex-, and race-specific attack rates. Although, true exposure is dependent on number of walking miles, these data do not exist. However, lack of these denominator data should not be used as an excuse for inaction (3).

Many Alaska Natives, particularly in Anchorage, do not own cars and are dependent on their feet for transportation. The Alaska Native Medical Center is located in the heart of downtown, near the intersection of the Glenn Highway and the Seward Highway. In addition, major innercity arterials of 4th, 5th, 6th Avenues carry large numbers of fast moving motor vehicles in complicated traffic patterns. Numerous fatalities also have occurred in the Spenard area, adjacent to Minnesota Drive and Spenard Road—both carry large numbers of fast-moving motor vehicles. In both areas, lighting is poor and barriers between pedestrians and motor vehicles are minimal or nonexistent.

Unfamiliarity with fast-moving traffic, wearing dark clothes, walking along dimly lit main traffic routes, and unavailability of motorized transportation may all increase risk of pedestrian injury (4). Previous studies have documented widespread, high-frequency hearing loss among adult Alaskan Natives, especially males (5). Decreased hearing may also increase pedestrian risk (6). Individuals who have never been licensed drivers may be less aware of the margins of safety needed by pedestrians to avoid motor vehicles. Many pedestrians who have not grown up in urban, heavily-trafficked environments have no understanding or appreciation of traffic-control devices such as signal lights and pedestrian-crossing signals (6).

Increased emphasis needs to be placed on improving physical separation of pedestrians and motor vehicles, increasing street lighting, increasing visibility of pedestrians by encouraging use of light-colored clothing or reflector strips on clothes, and increasing enforcement of speed limits.

A new Alaska Native Medical Center that will handle over one million out-patient visits per year is to be constructed at Bragaw Street and Tudor Road, scheduled for completion by 1994. Tudor Road is one of the most heavily travelled roads in Alaska, has a speed limit of 50 mph, has five lanes with a middle left turn and merge lane, and is poorly lit. Every effort needs to be made *now* by the Indian Health Service to insist that project designers and traffic engineers incorporate all possible features to enhance pedestrian safety. Discussion also need to be initiated with state and local traffic safety experts to review traffic flow patterns to assure pedestrian safety.

Elderly individuals also are overrepresented as fatalities both in national statistics and in Alaskan statistics. The elderly are less able to recover from a similar degree of injury than the young. The elderly also have decreased speed of walking, decreased agility, decreased vision, and decreased hearing , all of which impair ability to detect and avoid oncoming motor vehicles (7).

Avoidance of pedestrian fatalities is not easily achieved by focusing only on the driver (8). In five years, 94 pedestrians were killed in 92 crash events. In Alaska, there averaged 300,000 licensed drivers per year during the period 1980-1984. One fatal pedestrian collision occurred for every 3,250 licensed drivers. Current data systems are too insensitive to define accurately risk factors to identify drivers at highest risk of being involved in pedestrian fatal events.

We must increase our efforts to change behavior—of both pedestrians and drivers. But, decades of experience have taught that our ability to modify behavior is limited (8). While we continue efforts to reduce alcohol use among pedestrians and drivers, we must also increase efforts to reduce pedestrian fatalities by modifying the environment to separate pedestrians from motor vehicles. We must use existing knowledge, technology, and analysis of our present experience to modify the environment. We should increase our surveillance of injuries and fatalities. Our study focused only on pedestrian fatalities. No on-going surveillance systems exist to collect information on injuries in Alaska.

When pedestrian fatalities occur we should investigate each incident using a multi-disciplinary team, including engineers, traffic control specialists, and health professionals, in order to determine risk factors and, whenever possible, modify or eliminate them. Increased efforts need to be directed at modifying the environment to protect pedestrians from motor vehicles. Particularly in Anchorage efforts need to be directed to lower pedestrian fatalities among Adult Alaskan Natives.

ACKNOWLEDGMENTS

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News from the American Society for Circumpolar Health

IUCH/WHO Circumpolar Conference on Tobacco and Health*

*Yellowknife, Northwest Territories, Canada, April 4-6, 1989. Organized by International Union for Circumpolar Health, World Health Organization, Regional Office for Europe, and Department of Health, Government of the Northwest Territories, Canada.

Recommendations

The increasing use of tobacco products in the circumpolar area is a serious health problem. In this area there are extremely high proportions of smokers both in females and males, there is an alarming common use of smokeless tobacco, even among school children, and a steep increase in smoking related diseases, especially lung cancer. The following recommendations for regional action are made against the background of general acceptance of the WHO strategy for tobacco control, and have been approved at the Conference in order to set up an anti-tobacco policy in the area.

1. Elimination of the use of tobacco should be a prime concern of local health promotion agencies and regional health boards together with elimination of other self destructive behaviors such as alcohol and drug abuse.
2. Health promotion and education on all forms of tobacco use (smokeless tobacco included) should be strengthened, focusing on children, adolescents, pregnant women and socially and economically less fortunate people.
    - Use mainly visualized material!
    - Use material of local relevance!
    - Use the local language!
    - Use simple and understandable terms!
    - Utilize peer group support to encourage young people and adolescents not to use tobacco!
    - Include elders and youth representatives in conferences on tobacco and health!
    - Use native role models in public information campaigns!
3. Physicians and other health professionals should deliver anti-tobacco messages.
4. All politicians, civil servants, teachers, clergymen, physicians and other health professionals, and parents should set an example by not smoking!
5. Involve local people through regional health boards, voluntary organizations and consumer's groups in order to define local tobacco policies and take action to influence local and national authorities.
6. Promote and extend tobacco-free environments (schools, workstation, health facilities, banks, public transport).
7. Sale of tobacco to children should be banned. These bans should be strictly enforced with severe penalties for violation. Sale of broken packs should be forbidden.
8. Collect on-going local statistics on tobacco use and tobacco related diseases in cooperation with community representative, and make the results available to the population.
9. Prohibit all advertising and promotion of tobacco products, including sponsorship of sports and cultural events.
10. Increase taxes on tobacco products and allocate a portion of tobacco tax revenue to health promotion activities.
11. All tobacco products should carry explicit, prominent, and clearly visible rotating warnings about health risks.
12. Encourage physicians and researchers to make their research results available to the indigenous people.

Reported by Dr. J. P. Hart Hansen
President, International Union for Circumpolar Health

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ALASKA
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News from the Alaska Public Health Association

President's Message

The 14th Annual Health Congress held in Anchorage on April 24 -26, 1989 reflected many of the services and studies throughout the state. The problems and plans of the past, present and future were presented and discussed. Of special interest were the speakers on the opening day. Phillip W. Ulmer, PE Safety and Health Director for ARCO Alaska, Inc., discussed the need for more involvement by public health in occupational settings. Myra M. Munson, M.S.W., J.D. Commissioner, Department of Health and Social Services for the State of Alaska, spoke to the continued need for the development of a state health policy and the problems involved in the state's participation in the policy development. Commissioner Munson outlines these problem areas:

1. the administration will be in transition with the election of a new governor, thus a new administration may not have a continued commitment to the health policy development,
2. "no extra bodies" are available to work on the development of a state health policy. The person that has been assigned to look at development of a policy is already fully involved in other work duties, and,
3. major project, i.e. the Healthy Baby Project, takes precedent over planning.

Commissioner Munson again challenged the attendees at the congress to lead an advocacy group composed of professionals and the public to show the new administration that a Public Health Policy is needed.

Elizabeth Ward, RN, MN, Director, Division of Health and Social Services reported that there is good news from the Division of H&SS. Modern management is now in place and there is a cohesiveness among the departments. The leading areas of concern are prenatal care, assessment of health needs through data collection, and the Prince William Sound oil disaster. Director Ward talked specifically to the needs of the high risk teens and poor who are children. Complicating the service to the needy is the political message from Alaskans to get rid of more state workers. Director Ward discussed the difficult task of planning, funding and delivering services in the midst of this political climate.

The rest of the congress was filled with excellent speakers from Barrow to Honolulu. There was a wide range of topics and discussion. During the ALPHA annual business meeting, five resolutions were passed (see following pages).

The coming year will be an exciting and eventful time. Your participation is vital for ALPHA to accomplish its goals.

1. To increase membership to 225 members. What can you do? Maintain your membership, invite a friend to join, or invite three to join. Our membership as of April 24 was 133 members.
2. To increase the attendance at the Brown Bag Lunches to 700 (statewide) and the sites to 7.
3. Advocacy for various public health legislation. When you receive a letter from the ALPHA Health Issues Committee asking you to call or send a POM to your local legislators, do it quickly and have others in your area do the same.
4. Advocacy for the Development of a State Public Health Policy by May 1990. Contact Brian Saylor (Pres. elect) who is leading a group of interested public health providers, and planners in developing a strategic plan to advocate for the development of a state public health policy.

These are just a few ways that you can become more involved in the expansion of ALPHA and the implementation of the yearly goals. It is your continued support and active participation that makes the organization a vital force in Alaska. I call upon each member to become more active than last year and plan to hear from each of you.

Susan Jones, RN, MN
President
Alaska Public Health Association

RESOLUTIONS ADOPTED AT THE 14TH ANNUAL HEALTH CONGRESS

RESOLUTION 89-1

SUBJECT: Community Health Aide Program Funding

The Alaska Public Health Association,

Recognizing that the Community Health Aide Program (CHAP) is the backbone of the entire native health care system in Alaska*, and

Knowing that more than 40,000 rural Alaska Natives living in isolated villages depend on the CHAP as their sole means for primary health care and access to secondary and tertiary care*, and

Believing that the CHAP is in crisis due to severe underfunding*, ** and

Understanding that the current fiscal year 1989 funding level of 45% of need is insufficient to sustain a quality health care system for rural Native Alaskans***, and

Noting that $16.7 million more dollars are needed annually to adequately fund the program to provide reasonable work schedules and salaries, training, support, and supervision to Community Health Aides; therefore,

1. Encourages the Alaska legislative delegation to support full funding for the CHAP, specifically for the FY90 year at $28.7 million by introducing legislation to Congress asking for $16.7 million to be added as a congressional line item to the CHAP..
2. Encourage Congress to appropriate the $16.7 million needed to strengthen this critical program.

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* Caldera, D.L.: Alaska Community Health Aide Program In Crisis. Presented to Congress, March 1988.

** Caldera, D.L.: Alaska Community Health Aide Program in Crisis: A Briefing Paper. Presented to Congress, March 1988.

***Caldera, D.L.: Community Health Aide Program: The Crisis Remains. Presented to Congress, March 1989.

RESOLUTION 89-2

SUBJECT: Access to Health Care for All Alaskans

The Alaska Public Health Association,

Believing that access to health care is a right for all citizens, and

Finding that there are at least 40,000 Alaskans who do not have financial access to health care (1), and

Recognizing that a lack of access to public health and primary medical care due to either inadequate delivery systems or poor financing of care will create greater illness and increased costs of care to the individual and society later, (1,3) and

Noting that health care accounts for only four percent of all state and local government spending in Alaska compared to an average for all states of about 12%, (1, p.1) and

Further noting that the economy of the State of Alaska, and the resulting unemployment, have greatly increased barriers to financial access to health care on the part of many individuals, especially the working poor and/or uninsured, the uninsurable and the elderly, (3), therefore,

Commends the Governor of Alaska, the Commissioner of Health and Social Services and the Legislature for their leadership in establishing and supporting the work of the Governor's Interim Commission of Health Care and preparing an excellent and timely report of their work, *"The Best of Care: The Challenge of Providing Health Care to Alaskans."* The ALPHA Health Issues Committee should evaluate the report and make recommendations to the board. The board is empowered to transmit those recommendations to the governor and the legislature.

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1. Governor's Interim Commission on Health Care: *The Best of Care* Sept. 1988..
2. American Hospital Association: *Medicaid Options: State Opportunities and Strategies for Expanding Eligibility* 1987.
3. Sisters of Providence: "Uncompensated Care -- Patients Who Cannot Pay--Threatening the Future of Alaskan Health Care", Unpublished paper, January, 1989.

RESOLUTION 89-3
SUBJECT: State Public Health Policy Development

The Alaska Public Health Association,

Whereas the State of Alaska has no stated public health policy, and

Whereas such a policy is of critical importance to the development of the future of public health in our state given the unclear status of future resources, and

Recognizing that such a policy to be meaningful has to have the support of the legislative and executive branches of state government, and other important health policy groups in the state, therefore

Calls upon the Governor and legislature through the Commissioner of Health to lead the effort to develop a state health policy. Among other features:

- insures that basic public health issues which have demonstrated a significant historical impact are available to every resident of the state,
- reduces the present incidence of disease, disability, and death where there is a demonstrated potential to do so,
- insures that every resident of the state has access to quality health care at reasonable cost,
- promotes the occupational health, and
- protects and improves the environmental quality of the state.

Fiscal Impact on ALPHA: This resolution has no direct fiscal impact on ALPHA.

Public Health Impact: An agreed upon public health policy statement in Alaska would provide for a reasoned deployment of scarce resources to protect and promote fundamental public health in our state.

Calls upon the government and legislature during the interim to continue funding levels necessary to support public health services considered essential for the protection of the public health and welfare.

RESOLUTION 89-4
SUBJECT: Waste Management

The Alaska Public Health Association,

Recognizes that waste management is becoming a personal and environmental health problem in Alaska and in the world, and

Recognizes that every citizen produces an average of five pounds of refuse each day and that over 80% of our solid waste is dumped in landfills, and

Recognizes that we must develop a unified view of general and hazardous waste management so as to avoid control methods that simply shift pollution from one medium (air, land, water) to another, and

Recognizes that our disposal capacity for waste is becoming more limited as our population grows, and

Recognizes that general waste within very limited local landfills are often located close to the community, near natural waterways, and/or over permafrost locked land, and

Recognizes the high cost to the individual, business, and community to remove both volumes of trash and quantities of hazardous waste from Alaskan communities, and

Recognizes that the Congress of the United States, in adopting the Hazardous and Solid Waste Amendments in 1984, established a national policy that wherever feasible, the generation of hazardous waste should be reduced or eliminated as expeditiously as possible, and

Recognizes that hazardous waste reduction can improve workplace safety as well as lower waste management and regulatory costs, and

Recognizes that the mere disposal of waste is not the answer to its proper management, therefore

Recommends that the State of Alaska

- take an active role in protecting its residents, visitors, communities, and natural environment through an aggressive waste management program that will include:
- prioritization of waste management practices in the following order;
    1) waste source reduction,
    2) recycling of waste,
    3) waste treatment,
    4) waste disposal, and
- Provide leadership for all local governments and Alaskan business through setting the example of generating less waste, requiring recycling in all

state activities, requiring that the State, in concert with local governments and contractors, follow a recent federal decision requiring requiring federal agencies to alter procurement practices to encourage the purchase of re-refined petroleum products, and that this concept be expanded to paper, glass, aluminum, and other recycled products, and

- provide incentives for research into waste reduction ideas through a system of matching grants both to private businesses and independent researchers, and
- provide technical and educational assistance to rural communities to enable them to reduce waste generation, and recycle community waste cooperatively with State waste management efforts to reap greater cost benefits to all, and
- Look to a new philosophy of doing business that will have less impact on the environment including the use and development of renewable resources with the overall reduction of waste

RESOLUTION 89 - 5
SUBJECT: **Dental Care for Pre School Children in Rural Alaska**

The Alaska Public Health Association,

Recognizing that dental care especially at an early age can greatly improve oral health, and

Noting that there is currently a limited quantity and variety of dental services available in rural Alaska, and

Realizing that the preschool age population is increasing, therefore,

1. Commands the oral health survey conducted by Rural Cap and the Alaska Area Native Health Services on the identification of the dental treatment needs and costs of preschool age children in the State ofAlaska.
2. Encourages continuing research into the needs and costs of improved oral health for preschool children, and.
3. Recommends continued federal support and interagency cooperation in establishing an improved dental delivery system for preschool age children.

ALASKA PUBLIC HEALTH ASSOCIATION ANNUAL AWARDS

Barbara Berger Award - Bert Hall
Alaska Meritorious Health Service Award - Walter Johnson
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Contraindications: VASOTEC® (Enalapril Maleate, MSD) is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor.

Warnings: *Angioedema.* Angioedema of the face, extremities, lips, tongue, glottis, and/or larynx has been reported in patients treated with ACE inhibitors, including VASOTEC. In such cases, VASOTEC should be promptly discontinued and the patient carefully observed until the swelling disappears. In instances where swelling has been confined to the face and lips, the condition has generally resolved without treatment, although antihistamines have been useful in relieving symptoms. Angioedema associated with laryngeal edema may be fatal. **Where there is involvement of the tongue, glottis, or larynx likely to cause airway obstruction, appropriate therapy, e.g., subcutaneous epinephrine solution 1:1000 (0.3 mL to 0.5 mL), should be promptly administered.** (See ADVERSE REACTIONS.)

Hypotension: Excessive hypotension is rare in uncomplicated hypertensive patients treated with VASOTEC alone. Heart failure patients given VASOTEC commonly have some reduction in blood pressure, especially with the first dose, but discontinuation of therapy for continuing symptomatic hypotension usually is not necessary when dosing instructions are followed; caution should be observed when initiating therapy (See DOSAGE AND ADMINISTRATION.) Patients at risk for excessive hypotension, sometimes associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death, include those with the following conditions or characteristics: heart failure, hyponatremia, high-dose diuretic therapy, recent intensive diuresis or increase in diuretic dose, renal dialysis, or severe volume and/or salt depletion of any etiology. It may be advisable to eliminate the diuretic (except in heart failure patients), reduce the diuretic dose, or increase salt intake cautiously before initiating therapy with VASOTEC in patients at risk for excessive hypotension who are able to tolerate such adjustments. (See PRECAUTIONS, *Drug Interactions* and ADVERSE REACTIONS.) In patients at risk for excessive hypotension, therapy should be started under very close medical supervision and such patients should be followed closely for the first two weeks of treatment and whenever the dose of enalapril and/or diuretic is increased. Similar considerations may apply to patients with ischemic heart disease or cardiovascular disease in whom an excessive fall in blood pressure could result in a myocardial infarction or cerebrovascular accident. If excessive hypotension occurs, the patient should be placed in supine position and, if necessary, receive an intravenous infusion of normal saline. A transient hypotensive response is not a contraindication to further doses of VASOTEC, which usually can be given without difficulty once the blood pressure has stabilized. If symptomatic hypotension develops, a dose reduction or discontinuation of VASOTEC or concomitant diuretic may be necessary.

Neutropenia/Agranulocytosis: Another ACE inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression, rarely in uncomplicated patients but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease. Available data from clinical trials of enalapril are insufficient to show that enalapril does not cause agranulocytosis at similar rates. Foreign marketing experience has revealed several cases of neutropenia or agranulocytosis in which a causal relationship to enalapril cannot be excluded. Periodic monitoring of white blood cell counts in patients with collagen vascular disease and renal disease should be considered

Precautions: *General: Impaired Renal Function:* As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including VASOTEC, may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death.

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine were observed in 20% of patients. These increases were almost always reversible upon discontinuation of enalapril and/or diuretic therapy. In such patients, renal function should be monitored during the first few weeks of therapy.

Some patients with hypertension or heart failure with no apparent preexisting renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when VASOTEC has been given concomitantly with a diuretic. This is more likely to occur in patients with preexisting renal impairment. Dosage reduction and/or discontinuation of the diuretic and/or VASOTEC may be required.

Evaluation of patients with hypertension or heart failure should always include assessment of renal function. (See DOSAGE AND ADMINISTRATION.)

Hyperkalemia: Elevated serum potassium (> 5.7 mEq/L) was observed in approximately 1% of hypertensive patients in clinical trials. In most cases these were isolated values which resolved despite continued therapy. Hyperkalemia was a cause of discontinuation of therapy in 0.28% of hypertensive patients. In clinical trials in heart failure, hyperkalemia was observed in 3.8% of patients, but was not a cause for discontinuation.

Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with VASOTEC. (See *Drug Interactions*.)

Surgery/Anesthesia: In patients undergoing major surgery or during anesthesia with agents that produce hypotension, enalapril may block angiotensin II formation secondary to compensatory renin release. If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion.

Information for Patients:

Angioedema: Angioedema, including laryngeal edema, may occur especially following the first dose of enalapril. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to take no more drug until they have consulted with the prescribing physician

Hypotension: Patients should be cautioned to report lightheadedness especially during the first few days of therapy. If actual syncope occurs, the patients should be told to discontinue the drug until they have consulted with the prescribing physician.

All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduction in fluid volume. Other causes of volume depletion such as vomiting or diarrhea may also lead to a fall in blood pressure; patients should be advised to consult with the physician.

Hyperkalemia: Patients should be told not to use salt substitutes containing potassium without consulting their physician.

Neutropenia: Patients should be told to report promptly any indication of infection (e.g., sore throat, fever) which may be a sign of neutropenia

NOTE. As with many other drugs, certain advice to patients being treated with enalapril is warranted. This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects

Drug Interactions.

Hypotension: Patients on *Diuretic Therapy:* Patients on diuretics and especially those in whom diuretic therapy was recently instituted may occasionally experience an excessive reduction of blood pressure after initiation of therapy with enalapril. The possibility of hypotensive effects with enalapril can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with enalapril. If it is necessary to continue the diuretic, provide close medical supervision after the initial dose for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and DOSAGE AND ADMINISTRATION.)

Agents Causing Renin Release. The antihypertensive effect of VASOTEC is augmented by antihypertensive agents that cause renin release (e.g., diuretics).

Other Cardiovascular Agents: VASOTEC has been used concomitantly with beta-adrenergic-blocking agents, methyldopa, nitrates, calcium-blocking agents, hydralazine, prazosin, and digoxin without evidence of clinically significant adverse interactions

Agents Increasing Serum Potassium: VASOTEC attenuates potassium loss caused by thiazide-type diuretics. Potassium-sparing diuretics (e.g., spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium. Therefore, if concomitant use of these agents is indicated because of demonstrated hypokalemia, they should be used with caution and with frequent monitoring of serum potassium. Potassium-sparing agents should generally not be used in patients with heart failure receiving VASOTEC

Lithium: A few cases of lithium toxicity have been reported in patients receiving concomitant VASOTEC and lithium and were reversible upon discontinuation of both drugs. Although a causal relationship has not been established, it is recommended that caution be exercised when lithium is used concomitantly with VASOTEC and serum lithium levels should be monitored frequently

Pregnancy—Category C: There was no fetotoxicity or teratogenicity in rats treated with up to 200 mg/kg/day of enalapril (333 times the maximum human dose). Fetotoxicity, expressed as a decrease in average fetal weight, occurred in rats given 1200 mg/kg/day of enalapril but did not occur when these animals were supplemented with saline. Enalapril was not teratogenic in rabbits. However, maternal and fetal toxicity occurred in some rabbits at doses of 1 mg/kg/day or more. Saline supplementation prevented the maternal and fetal toxicity seen at doses of 3 and 10 mg/kg/day, but not at 30 mg/kg/day (50 times the maximum human dose).

Radioactivity was found to cross the placenta following administration of labeled enalapril to pregnant hamsters.

There are no adequate and well-controlled studies in pregnant women. VASOTEC® (Enalapril Maleate, MSD) should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Nursing Mothers: Milk in lactating rats contains radioactivity following administration of ^{14}C enalapril maleate. It is not known whether this drug is secreted in human milk. Because many drugs are secreted in human milk, caution should be exercised when VASOTEC is given to a nursing mother.

Pediatric Use: Safety and effectiveness in children have not been established.

Adverse Reactions: VASOTEC has been evaluated for safety in more than 10,000 patients, including over 1000 patients treated for one year or more. VASOTEC has been found to be generally well tolerated in controlled clinical trials involving 2987 patients.

Hypertension: The most frequent clinical adverse experiences in controlled trials were: headache (5.2%), dizziness (4.3%), and fatigue (3%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in controlled clinical trials were: diarrhea (1.4%), nausea (1.4%), rash (1.4%), cough (1.3%), orthostatic effects (1.2%), and asthenia (1.1%).

Heart Failure: The most frequent clinical adverse experiences in both controlled and uncontrolled trials were: dizziness (7.9%), hypotension (6.7%), orthostatic effects (2.2%), syncope (2.2%), cough (2.2%), chest pain (2.1%), and diarrhea (2.1%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in both controlled and uncontrolled clinical trials were: fatigue (1.8%), headache (1.8%), abdominal pain (1.6%), asthenia (1.6%), orthostatic hypotension (1.6%), vertigo (1.6%), angina pectoris (1.5%), nausea (1.3%), vomiting (1.3%), bronchitis (1.3%), dyspnea (1.3%), urinary tract infection (1.3%), rash (1.3%), and myocardial infarction (1.2%).

Other serious clinical adverse experiences occurring since the drug was marketed or adverse experiences occurring in 0.5% to 1% of patients with hypertension or heart failure in clinical trials in order of decreasing severity within each category:

Cardiovascular: Myocardial infarction or cerebrovascular accident, possibly secondary to excessive hypotension in high-risk patients (see WARNINGS, *Hypotension*); cardiac arrest, pulmonary embolism and infarction, rhythm disturbances; atrial fibrillation, palpitation

Digestive: Ileus, pancreatitis, hepatitis or cholestatic jaundice, melena, anorexia, dyspepsia, constipation, glossitis.

Nervous/Psychiatric: Depression, confusion, ataxia, somnolence, insomnia, nervousness, paresthesia.

Urogenital: Renal failure, oliguria, renal dysfunction (see PRECAUTIONS and DOSAGE AND ADMINISTRATION), prostate hypertrophy.

Respiratory: Bronchospasm, rhinorrhea, asthma, upper respiratory infection.

Skin: Herpes zoster, pruritus, alopecia, flushing, photosensitivity.

Other: Muscle cramps, hyperhidrosis, impotence, blurred vision, taste alteration, tinnitus.

A symptom complex has been reported which may include fever, myalgia, and arthralgia, an elevated erythrocyte sedimentation rate may be present. Rash or other dermatologic manifestations may occur. These symptoms have disappeared after discontinuation of therapy

Angioedema: Angioedema has been reported in patients receiving VASOTEC (0.2%). Angioedema associated with laryngeal edema may be fatal. If angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with VASOTEC should be discontinued and appropriate therapy instituted immediately (See WARNINGS.)

Hypotension: In the hypertensive patients, hypotension occurred in 0.9% and syncope occurred in 0.5% of patients following the initial dose or during extended therapy. Hypotension or syncope was a cause for discontinuation of therapy in 0.1% of hypertensive patients. In heart failure patients, hypotension occurred in 6.7% and syncope occurred in 2.2% of patients. Hypotension or syncope was a cause for discontinuation of therapy in 1.9% of patients with heart failure (See WARNINGS.)

Clinical Laboratory Test Findings:

Serum Electrolytes: Hyperkalemia (see PRECAUTIONS), hyponatremia.

Creatinine, Blood Urea Nitrogen: In controlled clinical trials, minor increases in blood urea nitrogen and serum creatinine, reversible upon discontinuation of therapy, were observed in about 0.2% of patients with essential hypertension treated with VASOTEC alone. Increases are more likely to occur in patients receiving concomitant diuretics or in patients with renal artery stenosis. (See PRECAUTIONS.) In patients with heart failure who were also receiving diuretics with or without digitalis, increases in blood urea nitrogen or serum creatinine, usually reversible upon discontinuation of VASOTEC and/or other concomitant diuretic therapy, were observed in about 11% of patients. Increases in blood urea nitrogen or creatinine were a cause for discontinuation in 1.2% of patients.

Hemoglobin and Hematocrit: Small decreases in hemoglobin and hematocrit (mean decreases of approximately 0.3 g % and 1.0 vol %, respectively) occur frequently in either hypertension or heart failure patients treated with VASOTEC but are rarely of clinical importance unless another cause of anemia coexists. In clinical trials, less than 0.1% of patients discontinued therapy due to anemia.

Other (Causal Relationship Unknown): In marketing experience, rare cases of neutropenia, thrombocytopenia, and bone marrow depression have been reported.

Liver Function Tests: Elevations of liver enzymes and/or serum bilirubin have occurred.

Dosage and Administration: *Hypertension:* In patients who are currently being treated with a diuretic, symptomatic hypotension occasionally may occur following the initial dose of VASOTEC. The diuretic should, if possible, be discontinued for two to three days before beginning therapy with VASOTEC to reduce the likelihood of hypotension. (See WARNINGS.) If the patient's blood pressure is not controlled with VASOTEC alone, diuretic therapy may be resumed

If the diuretic cannot be discontinued, an initial dose of 2.5 mg should be used under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions*.)

The recommended initial dose in patients not on diuretics is 5 mg once a day. Dosage should be adjusted according to blood pressure response. The usual dosage range is 10 to 40 mg per day administered in a single dose or in two divided doses. In some patients treated once daily, the antihypertensive effect may diminish toward the end of the dosing interval. In such patients, an increase in dosage or twice-daily administration should be considered. If blood pressure is not controlled with VASOTEC alone, a diuretic may be added.

Concomitant administration of VASOTEC with potassium supplements, potassium salt substitutes, or potassium-sparing diuretics may lead to increases of serum potassium (see PRECAUTIONS).

Dosage Adjustment in Hypertensive Patients with Renal Impairment: The usual dose of enalapril is recommended for patients with a creatinine clearance >30 mL/min (serum creatinine of up to approximately 3 mg/dL). For patients with creatinine clearance ≤30 mL/min (serum creatinine ≥3 mg/dL), the first dose is 2.5 mg once daily. The dosage may be titrated upward until blood pressure is controlled or to a maximum of 40 mg daily.

Heart Failure: VASOTEC is indicated as adjunctive therapy with diuretics and digitalis. The recommended starting dose is 2.5 mg once or twice daily. After the initial dose of VASOTEC, the patient should be observed under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions*.) If possible, the dose of the diuretic should be reduced, which may diminish the likelihood of hypotension. The appearance of hypotension after the initial dose of VASOTEC does not preclude subsequent careful dose titration with the drug, following effective management of the hypotension. The usual therapeutic dosing range for the treatment of heart failure is 5 to 20 mg daily given in two divided doses. The maximum daily dose is 40 mg. Once-daily dosing has been effective in a controlled study, but nearly all patients in this study were given 40 mg, the maximum recommended daily dose, and there has been much more experience with twice-daily dosing. In addition, in a placebo-controlled study which demonstrated reduced mortality in patients with severe heart failure (NYHA Class IV), patients were treated with 2.5 to 40 mg per day of VASOTEC, almost always administered in two divided doses. (See CLINICAL PHARMACOLOGY, *Pharmacodynamics and Clinical Effects*.) Dosage may be adjusted depending upon clinical or hemodynamic response. (See WARNINGS.)

Dosage Adjustment in Heart Failure Patients with Renal Impairment or Hyponatremia: In heart failure patients with hyponatremia (serum sodium <130 mEq/L) or with serum creatinine >1.6 mg/dL, therapy should be initiated at 2.5 mg daily under close medical supervision. (See DOSAGE AND ADMINISTRATION, *Heart Failure*, WARNINGS, and PRECAUTIONS, *Drug Interactions*.) The dose may be increased to 2.5 mg b.i.d., then 5 mg b.i.d. and higher as needed, usually at intervals of four days or more, if at the time of dosage adjustment there is not excessive hypotension or significant deterioration of renal function. The maximum daily dose is 40 mg

For more detailed information, consult your MSD representative or see Prescribing Information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, PA 19486. J6VS18R(815)

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History of Medicine in Alaska

FRANCIS J. PHILLIPS, M.D., 1904 - 1989

Francis Phillips was born in 1904 a farm boy near Hay Springs, Nebraska. He was the youngest of five by seven years.

His father, Joseph Phillips, left Oxford, Wisconsin at the age of 14 because the family was so big that the children did not have much to eat. His oldest sister loaned him fifty dollars to buy a wagon and two horses. He started out with his cousin to go west. In Omaha, his cousin induced him to stop for a beer. But when the cousin stayed for another, Joseph Phillips drove on alone to homestead in Preston, Iowa.

When the railroad came within 100 miles, he felt this too encroaching, so he moved westward. In Nebraska, he found an abandoned homestead with a good well which he claimed. Within three years he had twenty acres under cultivation.

In 1891, Annis Hidlay came to Hay Springs to live with her sister. Annis and Joseph Phillips met and were married.

His mother was small and rugged. She was intelligent but had quit school in the eighth grade. Francis Phillips recalls writing her from college, deliberately using big words to confound her, only to have her purchase a dictionary to keep up with him.

His mother made butter and raised chickens. She would take these to town some four miles away, delivering the items to individual homes. When Francis was seven, his father died, but his mother carried on valiantly.

Francis Phillips greatly admired his mother who died at the age of ninety. During her last illness, when her family became too solicitous, she was heard to say: "Will you kindly mind your own business and let me get on with dying."

Francis attended a country school until the eighth grade. Then he had to ride a horse into Hay Springs. He remembers one morning after a heavy snow getting up an hour earlier in order to walk. It was impossible to see the road or even the fences, but he got to school.

As a boy, he trapped muskrats which brought 85 cents a skin. During World War I, despite his age of barely thirteen, he filled in on the farms because so many men had been drafted. Later, he worked on a threshing crew at $1.25 per week plus room. He found himself in crews of huge men of Swedish and Bohemian decent. He was always the smallest and was teased for it, but he learned to program his labor and especially how to work with both hands.

In high school, he curried and harnessed horses to earn money. However, Francis decided that he must continue in school, because he was awfully ignorant.

The Normal School (teachers' college) was twenty miles away. His mother and he rented a house there. He took extra courses and worked after school. By the time he went to his freshman year at the University of Nebraska, Lincoln he had nineteen credits but the university allowed only six. He didn't understand their decision then or later.

It took over four years to earn a degree, because he majored in English literature and history and later decided to take premedical courses. For two years he graded papers in the English department which he found ironic. During the first six weeks at the university, he had been given the option of learning to spell or go home. Also he taught a class in English literature.

He went on to the University of Chicago to complete his premed. He took anatomy, dissecting a cadaver before medical school. However, the anatomy professor wanted him to do a research project. Because Francis Phillips did not like to be caught on anything, he went to the library and discovered that the suggested project had already been done. He flunked anatomy.

He also took embryology which excited him greatly. He did advanced work in embryology, waiting on tables to pay his way. He thought of a Ph.D. in embryology but was thwarted when he was prevented from getting a scholarship.

Next he found himself in chemistry. He worked as a deaner in the laboratory of the University of Chicago Clinic. He became intrigued with the problem of iodine analysis. So he spent about eight hours–a–day for a week reading in German all he could about it.

When George M. Curtis, M.D., a goiter surgeon interested in physiological chemistry, went to Ohio State University, Francis went along as a chemist. He set up and ran a laboratory on iodine research. In 1933, the chairman of the pharmacology section of the American Medical Association invited him to speak on iodine and goiter. He talked for six minutes. There was a 45 minute discussion. He was elected to Sigma Xi, an honorary science society.

Finally, he attended Rush Medical School. After receiving his M.D. in 1937, he had a 16–month internship at Presbyterian Hospital, Chicago: half medicine and half surgery.

Dr. Phillips considered entering general practice, but thoracic surgery was being developed at the University of Chicago. He became a thoracic surgery resident there for one year, followed by a year at a tuberculosis sanitorium in Northville, Michigan, and an 11–month appointment as chief resident at Herman Keifel Hospital, Detroit, a large contagious disease hospital. He recalls that in his last week there, he performed 11 major and 27 minor surgeries.

He was then called into the Army. Serving four-and-a-half years during World War II, his entire practice consisted of wiring one knee cap at Atka and applying one first aid dressing in Europe.

He was stationed in Atka for five months. The hospital was three large quonset huts. Its chief was a specialist in tropical medicine. He might have made reports to Washington, D.C.: "No report of Anopheles mosquito on Atka." Dr. Phillips got mumps encephalitis while on Atka and was given typhoid vaccine to stimulate his white cells. Despite the treatment, he recovered.

After spending a month at home, he was sent with the 28th Infantry Division of the Pennsylvania National Guard to Wales, and on the eleventh day of the invasion, to Europe. He was at the Battle of the Bulge.

He became Division Surgeon in the Saar Valley for displaced persons camps where there were 73,000 Germans, Russians, Estonians, and Roumanians among others. He was responsible for supplies to the American, French, and German hospitals and for public health.

He prided himself that he could speak German. However, he found himself in a camp with mixed nationalities where the cooks wouldn't cook. The cooks were Russian. A displaced Russian colonel came and spoke to the cooks who suddenly began working. The colonel explained to Dr. Phillips that he had given the cooks three choices: to cook, to go to Siberia, or to be shot.

Finally, he was sent to Michigan to train for amphibious landings on the coast of China but the war ended.

Back at the University of Chicago, he was hired as an instructor in surgery, but he won't say who got instructed. He finished the residency he had left dangling at the start of the war.

He became medical director of the Morgan Heights Sanatorium at Marquette, Michigan; then on to the Barnwell Veterans Administration Tuberculosis Hospital in Temple, Texas, where he was chief of chest surgery. There he was told that he did not regiment well, so he resigned.

He went on to the Gowen Sanatorium and Clinic in Shreveport, Louisiana as a chest surgeon and made $50,000 a year for a while in private practice.

In 1950 he had an offer from the Wesleyan Tuberculosis Sanatorium in Seward. Since he had always wanted to come back to Alaska, he did and remained in Alaska ever after.

Francis Phillips found himself in disagreement with the treatment of tuberculosis in the 1950s. He thought the only useful technique was surgery and that treatment by bed rest was ineffectual and exceedingly depressing. In a sense, people were put to bed and left to recover. In some incidents, lead shot bags were placed around patient's chests to limit movement.

He encouraged patients with tuberculosis to be active. When they improved, he encouraged them to do more. Dr. Phillips' optimism, a lifelong part of him, must have been a great boost to his patients.

He provided a rehabilitation center where recovering patients could learn new things, ranging from skin sewing to shoe repair to store management. An old army warehouse was used for this purpose.

Especially he stressed education. He went to Juneau to get a teacher for his program. The Methodist women who ran the sanitorium felt it unthinkable that a person could recover from tuberculosis and go to school. However, he prevailed. A boy from Eek passed third and fourth grades and got well.

Dr. Phillips encouraged his staff to reflect a positive attitude. It was said that this was a hospital with cheerful patients.

Dr. Phillips also stressed a high protein diet for the patients, rather then relying so heavily on the high carbohydrate fare from the hospital garden. It made a difference.

To encourage patients to come for treatment, Dr. Phillips picked them by geographical location so that when they recovered they would encourage others to get well.

When Francis Phillips first came to Seward, the average patient stayed nearly three years. Seven years later when he left, the average stay was six to eight weeks. In 1955, chemotherapy had arrived which made an enormous difference in treating the disease.

While in Seward, Dr. Phillips took over the practice of Dr. Joseph Sheldon, a general practitioner, who left to take ophthalmology training. Dr. Phillips had 150 patients in the sanitorium and 35 each day in the downtown office. The latter practice upset the American Board of Thoracic Surgeons, but it was necessary in Alaska at that time.

Francis Phillips cites the example of an Athabascan from Nenana who had bilateral lower lobe cavities as well as a congenital heart defect. In a year, his cavities had cleared. He hitchhiked to the Mayo Clinic for heart surgery. Later, he went to college and today works for the Bureau of Indian Affairs.

When Dr. Phillips came to Anchorage in 1957, he held chest clinics and served as city health officer. He taught nurses in a program at the Anchorage Community College, training them as school and public health nurses. Later in his career in Anchorage, Dr. Phillips was active in the treatment and rehabilitation of alcoholics. When asked what he specialized in, he replied: "The skin and its contents."

In addition to his own achievements, recognized by an honorary degree from the University of Alaska, Anchorage, he has been the pater familias of a useful family. Francis Philips had married Mary Lee Parsons in 1940. They had grown up together in Hay Springs. Their first daughter, Jane, was born on December 9, 1941, two days after Pearl Harbor. Susan was born in 1947. She is a painter in Homer. Mary Lee Phillips was president of the Alaska State Medical Association Auxiliary. There are six grandsons. The oldest is a junior at Stanford.

Dr. Francis James Phillips died in his sleep on April 12, 1989.

Gwynneth Gminder Wilson
Alaska State Medical Association Auxiliary

AUTHOR'S NOTE: While Dr. Phillips enjoyed being interviewed for this article, he never saw it. I had arranged to take it to him on the morning of April 12th. Consequently, it has not been edited by him for the inevitable errors that occur in this type of oral history. I am sorry and sad.

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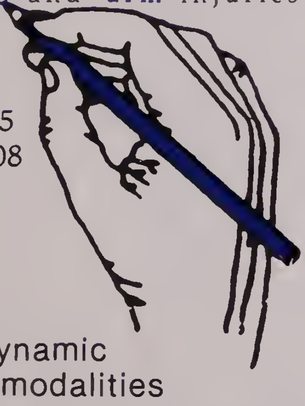

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Brief Summary

Consult the package literature for complete information.

Indications and Usage: Axid is indicated for up to eight weeks for the treatment of active duodenal ulcer. In most patients, the ulcer will heal within four weeks.

Axid is indicated for maintenance therapy for duodenal ulcer patients at a reduced dosage of 150 mg h.s. after healing of an active duodenal ulcer. The consequences of continuous therapy with Axid for longer than one year are not known.

Contraindication: Axid is contraindicated in patients with known hypersensitivity to the drug and should be used with caution in patients with hypersensitivity to other H_2-receptor antagonists

Precautions: *General* – 1. Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy

2. Because nizatidine is excreted primarily by the kidney, dosage should be reduced in patients with moderate to severe renal insufficiency

3. Pharmacokinetic studies in patients with hepatorenal syndrome have not been done. Part of the dose of nizatidine is metabolized in the liver. In patients with normal renal function and uncomplicated hepatic dysfunction, the disposition of nizatidine is similar to that in normal subjects.

Laboratory Tests – False-positive tests for urobilinogen with Multistix® may occur during therapy with nizatidine.

Drug Interactions – No interactions have been observed between Axid and theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin. Axid does not inhibit the cytochrome P-450-linked drug-metabolizing enzyme system; therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur. In patients given very high doses (3,900 mg) of aspirin daily, increases in serum salicylate levels were seen when nizatidine, 150 mg b.i.d., was administered concurrently

Carcinogenesis, Mutagenesis, Impairment of Fertility – A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups. The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a two-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny

Pregnancy – Teratogenic Effects – Pregnancy Category C – Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in one fetus and at 50 mg/kg it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in one fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers – Studies conducted in lactating women have shown that <0.1% of the administered oral dose of nizatidine is secreted in human milk in proportion to plasma concentrations. Caution should be exercised when administering nizatidine to a nursing mother.

Pediatric Use – Safety and effectiveness in children have not been established.

Use in Elderly Patients – Ulcer healing rates in elderly patients are similar to those in younger age groups. The incidence rates of adverse events and laboratory test abnormalities are also similar to those seen in other age groups. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Clinical trials of nizatidine included almost 5,000 patients given nizatidine in studies of varying durations. Domestic placebo-controlled trials included over 1,900 patients given nizatidine and over 1,300 given placebo. Among reported adverse events in the domestic placebo-controlled trials, sweating (1% vs 0.2%), urticaria (0.5% vs < 0.01%), and somnolence (2.4% vs 1.3%) were significantly more common in the nizatidine group. A variety of less common events was also reported; it was not possible to determine whether these were caused by nizatidine.

Hepatic – Hepatocellular injury, evidenced by elevated liver enzyme tests (SGOT [AST], SGPT [ALT], or alkaline phosphatase), occurred in some patients and was possibly or probably related to nizatidine. In some cases, there was marked elevation of SGOT, SGPT enzymes (greater than 500 IU/L) and, in a single instance, SGPT was greater than 2,000 IU/L. The overall rate of occurrences of elevated liver enzymes and elevations to three times the upper limit of normal, however, did not significantly differ from the rate of liver enzyme abnormalities in placebo-treated patients. All abnormalities were reversible after discontinuation of Axid.

Cardiovascular – In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in two individuals administered Axid and in three untreated subjects.

CNS – Rare cases of reversible mental confusion have been reported.

Endocrine – Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to Axid. Impotence and decreased libido were reported with equal frequency by patients who received Axid and by those given placebo. Rare reports of gynecomastia occurred

Hematologic – Fatal thrombocytopenia was reported in a patient who was treated with Axid and another H_2-receptor antagonist. On previous occasions, this patient had experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental – Sweating and urticaria were reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity – As with other H_2-receptor antagonists, rare cases of anaphylaxis following administration of nizatidine have been reported. Because cross-sensitivity in this class of compounds has been observed, H_2-receptor antagonists should not be administered to individuals with a history of previous hypersensitivity to these agents. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other – Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine administration have been reported.

Overdosage: Overdoses of Axid have been reported rarely. The following is provided to serve as a guide should such an overdose be encountered

Signs and Symptoms – There is little clinical experience with overdosage of Axid in humans. Test animals that received large doses of nizatidine have exhibited cholinergic-type effects, including lacrimation, salivation, emesis, miosis, and diarrhea. Single oral doses of 800 mg/kg in dogs and of 1,200 mg/kg in monkeys were not lethal. Intravenous median lethal doses in the rat and mouse were 301 mg/kg and 232 mg/kg respectively.

Treatment – To obtain up-to-date information about the treatment of overdose, a good resource is your certified regional Poison Control Center. Telephone numbers of certified poison control centers are listed in the *Physicians' Desk Reference (PDR)*. In managing overdosage, consider the possibility of multiple drug overdoses, interaction among drugs, and unusual drug kinetics in your patient.

If overdosage occurs, use of activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. Renal dialysis for four to six hours increased plasma clearance.

PV 2096 AMP [013089]

Additional information available to the profession on request.

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1. Data on file, Lilly Research Laboratories.

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GLIMPSES OF ALASKAN MEDICAL HISTORY

Edited by Robert Fortuine, M.D.

Koniag Healers and their Methods (1804-5)

Captain-Lieutenant Yurii Lisianskii came to Alaska at the age of 31 as skipper of the Russian naval vessel *Neva*, which together with the *Nadezhda* became from 1803 to 1806 the first Russian ships to circumnavigate the globe. The two vessels parted company in the Hawaiian Islands, and in July 1804 Lisianskii reached the Russian settlement at Kodiak. A message awaited him there from Chief Manager Alexandr Baranov of the Russian-American Company, who requested his immediate assistance in dealing forcibly with the Tlingit. After a brief stay for necessary repairs, Lisianskii sailed directly to Sitka, where his ship's guns were instrumental in destroying the Tlingit fort. Leaving Baranov and his men to erect their own fortifications at the site, he returned to Kodiak to spend the winter.

The following June he proceeded once more to Sitka, where Baranov had been able to come to an understanding of sorts with the Indians. After spending the summer at the new settlement, Lisianskii at last made the *Neva* ready for sea again and sailed home to St. Petersburg via Canton.

During his winter on Kodiak Island, Lisianskii carefully observed not only the Russian trading establishment there, but also the Koniag and their customs. He visited several outlying settlements on the island, where his insatiable curiosity allowed him to collect invaluable information on the life of the people, including their homes, dress, social customs, religion, and not least their healing practices.

"Of the different diseases to which these people are subject, the most common are, venereal, colds, consumption, itch, and ulcers. The two last are so unavoidable, that there is hardly a person to be found on the island without one or the other. They have three methods of cure; shamaning, cutting away the part affected and bleeding. I was told, that slight venereal taints are removed by means of some decoction, but that when the disease touches the nose, each nostril is pierced through, and sometimes the gristle itself. . . .

"While in the village I had an opportunity of witnessing a curious method of bleeding. A young woman performed this office of the surgeon. She first transpierced the vein of the arm with a copper instrument, which was far from being sharp. As she did not succeed in drawing blood the first time, she repeated the operation, when the blood gushed out in a stream. Though to me this was no very agreeable sight, the patient sat with the most perfect composure, which surprised me when I understood that he had never been bled in this manner before. . . .

"On the island of Kadiack, as through the whole of what we saw on the northwest coast of America, shamaning is held in great veneration. The professors of it are brought up to it from their infancy. They persuade the people, they have a correspondence with the devil, and can by his means foretell what will come to pass. They pretend that some children are doomed to become shamans; and that their destiny is made known by a dream. Though every shaman has in his practice some particular mummery, the foundation of the pretended art is the same in all. Their ceremony generally observed on these occasions, is this:--The skin of a seal or other animal being spread on the ground, in the middle of a barabara or elsewhere, and a vessel of water placed near it, the shaman enters, and, placing himself on the skin, takes off his ordinary dress, and puts on a *kamleyka*, turning the fore part behind. He then disguises himself with a wig of human hair, to which two feathers are attached, one on each side, to resemble horns. Opposite to him stands the person who wishes to consult him about his affairs. The question to be solved being stated, the shaman begins to sing, the company joining in the song by degrees, till it comes to the chorus, or rather a yell. During this incantation, the shaman makes the most frightful grimaces and twistings of the body, till at last he appears perfectly exhausted and falls to the ground. He falls, however, only to rise again; and he repeats this foolery several times before he gives the answer, which, in his trance, he pretends to have received from the evil spirit.

"The shamans are consulted also as physicians in dangerous cases, and are rewarded very handsomely if the patient recovers, but receive nothing if he dies. The mode of cure consists, in like manner, in incantations."

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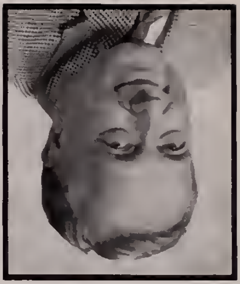

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Irvin A. Rothrock, M.D.
President, Alaska State Medical Association

Dear Fellow ASMA members,

The next issue of *Alaska Medicine* will probably carry Russ Huffman's picture on the president's page. I say probably because, as you are by now aware, John Middaugh has serious problems with visual perception which make it very difficult for him to identify the correct photograph of the president, and to properly spatially orient it. As a gesture of good will I have offered to help him pick out Russ's picture and to make certain he understands which is the top and the bottom of the picture. I hope that this will not place an undo strain upon his conceptual capacity.

Despite John's handicap, and one cannot hold him responsible for that, I suppose, he continues to produce our journal with ever increasing improvement in quality. He has also been a great deal of help to me throughout the past year. And so it has been with all the members of ASMA, the council, and with Ray Schalow. I only recently became active in medical politics, but I have been impressed with the members of this association who have been active for years and whose dedication and knowledge forms a reliable frame of reference for those of us, such as myself, who have more recently become involved in working actively in ASMA.

No one works harder than Ray Schalow. I know from comments that I hear, that many are still angry about the way Ray handled last year's financial crisis. First, let me emphasis that Ray did not create ASMA's financial problems, nor did these problems begin last year or the year before. The financial deficit which came to a head this past year had been building gradually for a number of years because we were simply spending more money then we were taking in. Ray's way of dealing with this may have not been the best, but in the past year of listening to, and participating in, debate regarding this problem I have not heard of a good way of dealing with it. Except one. The ultimate solution is to raise dues orcut services. Because this is a hard choice that no one wants to make, it has been postponed for several years. However, this year in Sitka we will have to decide what course we are going to pursue as an organization. The choice we make in Sitka will set the bounds of ASMA's operations for years to come.

ASMA's financial problem, although serious, was not the only issue which demanded attention this past year. No one, particularly those who were victims, will soon forget the Keystone Kops operation which combined the talents of the postal service and the Department of Labor in rounding up those physicians suspected of looting the public treasury by manipulating workers' compensation claims. If any action exemplifies the criticism that "a federal bureau can never constructively manage anything, it can only tyrannize," this caper certainly does. We have written our congressional delegations, and they have promised to investigate. We have not yet heard the outcome of their investigation.

The yearly council meeting in Juneau is the most direct political activity of this association. This is an expensive meeting. Is it worth it? Is a face-to-face discussion with a legislator more effective than a letter or a telephone call? I believe that it is, although I can not prove it. I believe that the meeting in Juneau is important, because although all legislators may not be, I believe that a great deal are sincerely interested in our concerns and want to hear from us. Perhaps this is a naive assumption on my part. Perhaps politicians are, as has been said, the only distinctive native American criminal class, and I am simply deluding myself when I think we can have any input unless we can assure the person of either money or votes. However, I choose not to believe this. I think we should continue the Juneau meeting because I think being there has an impact which is not possible to make by letter or phone call.

Working with ASMA, the council, and with Ray in trying to meet the challenges of the past year has not always been pleasant, but it has never been dull and I feel I have learned a lot.

All in all, it was a good year.

Irvin A. Rothrock
President
Alaska State Medical Association

> I'm glad that with his help we finally got Dr. Rockroth's picture right!
> —Editor

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My Turn

Jury Of My Peers

by S. Lon Conner[1]

Since the current malpractice litigation crisis began, personal injury lawyers have loudly insisted that the principal cause is the increasing number of incompetent or incapacitated physicians.

Just as insistently, organized medicine, from AMA down to the county society, has cited facts to support the counter-claim that, in general, the best doctors are the ones being sued.

Exhibit A here in Alabama could surely be the case of Howard C. Snider, Jr., M.D., Montgomery surgeon, and his senior partner at the time, John M. Cameron, M.D..

In the dozen years that I have been the Medical Association of the State of Alabama's executive director, upwards of 1,500 Alabama physicians have experienced the valley of death of a malpractice suit that hauled them before judge and jury or came close enough to bring terror to their hearts.

Unfortunately for Drs. Snider and Cameron, but fortunately for you, they were among those who experienced the bizarre and painful trial by jury and learned firsthand how facts are perverted and truth becomes an orphan of the storm. You are fortunate because Dr. Snider has written a book about his ordeal, *Jury of My Peers: A Surgeon's Encounter,* published by Penkevill Publishing Company.

Jury of My Peers is arguably the best book ever written on the subject by a physician victim of the malpractice mania. In fact, James S. Todd, M.D., Senior Executive Vice President of AMA, so argues. In a recent letter to Dr. Snider, Dr. Todd said: "You have captured better than any I know, the agonies and implications of our present system, with its damage to all involved."

Since I too was privileged to read the book before publication, I can happily second Dr. Todd's assessment, along with his statement that he simply could not put down *Jury of My Peers* until completion. It is a remarkably detailed and moving account of the malpractice plague.

Apart from medical papers, Dr. Snider had no previous experience as an author. But his mastery of detail, color, mood, and narrative technique is of such character as to suggest that in the crucible of his agony he was somehow invested with the power and skill to do what he felt he had to do to survive emotionally—tell the full, unvarnished story of his sudden descent into the madness of the tort system, where facts are manipulated or hidden, where truth is an enemy alien, and where the vaunted jury system is rendered absurd by proceedings that have become, in the span of a few years, a ridiculous mockery of our supposed age of scientific enlightenment.

Jury of My Peers should be read by every physician, of course; but it should also be read by every lawyer who plies his trade in the constant search for deep pockets, and by his fellow attorneys who are uncomfortable with this continuing abuse of process.

Because of the suspense in this book, and because I believe any summary would detract from the dramatic impact, I will not go into detail here.

Suffice it to say that the plaintiff was a woman with cancer so involved and so advanced that it might justifiably have been called inoperable. Dr. Cameron, assisted by Dr. Snider, operated anyway and their patient was cured by the heroic surgery Dr. Cameron is noted for.

But, like so many plaintiffs in malpractice cases, the patient's gratitude for deliverance from certain death turned into something entirely different as she brooded over side effects of the massive intervention.

While her life had been saved, she was not restored 100% to pre-cancer health—and that, as we have all learned to our consternation, is often the incredible basis for claims of negligence.

Thus began the nightmare that ultimately drove Dr. Cameron from practice and left Dr. Snider, even today, less than certain he will continue practice.

Drs. Cameron and Snider are sterling examples of physicians who have always sought the grail of perfection. Dr. Cameron, classmate of the esteemed John W. Kirklin, M.D., in the Harvard Medical Class of 1942, had long ago earned the reputation of one of the region's most gifted and dedicated surgeons.

It was Dr. Kirklin, in fact, advising Dr. Snider as a resident on his service, that he should try first to join Dr. Cameron's Montgomery group. Dr. Kirklin meant that as a double compliment—to his 1942 classmate and to young Dr. Snider, who clearly stood out as the kind of overachiever whose skills and personality would mesh with those of Dr. Cameron.

Dr. Snider had finished second in his medical school class, had been Phi Beta Kappa in undergraduate school, and named to honor societies from high school on through medical school.

One of the qualities that makes *Jury of My Peers* must reading for all physicians is the superb delineation of the trauma experienced by physicians trained in science and the quest for truth when they are thrown into the calculated madness of the civil justice system.

The culture shock of men and women disciplined in the search for truth as they are introduced to the travesty of that in tort court is surely one of the greater achievements of this book.

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[1] 2055 East South Boulevard, Suite 410, Montgomery, Alabama 36116.

If Dr. Snider's persuasive polemics against the system took up the entire 288 pages of *Jury of My Peers,* it would be well worth the reading for that alone. But there is so much more here: the devastating impact on practice and families, for example; the vast lottery of jury selection; the psychology of the physician under great emotional turmoil; even the relatively new concept of body language (which Dr. Snider, who earned a master's degree in counseling just to acquire this skill, applies with comic failure).

Jury of My Peers should now be available. It is absolutely must reading, cramming between two covers everything you need to know about the contagion that has preoccupied organized medicine for too many years.

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INDICATIONS AND USAGE: BECAUSE OF REPORTS OF INTESTINAL AND GASTRIC ULCERATION AND BLEEDING WITH SLOW-RELEASE POTASSIUM CHLORIDE PREPARATIONS, THESE DRUGS SHOULD BE RESERVED FOR THOSE PATIENTS WHO CANNOT TOLERATE OR REFUSE TO TAKE LIQUID OR EFFERVESCENT POTASSIUM PREPARATIONS OR FOR PATIENTS IN WHOM THERE IS A PROBLEM OF COMPLIANCE WITH THESE PREPARATIONS.

1 For therapeutic use in patients with hypokalemia with or without metabolic alkalosis, in digitalis intoxication and in patients with hypokalemic familial periodic paralysis

2 For the prevention of potassium depletion when the dietary intake is inadequate in the following conditions Patients receiving digitalis and diuretics for congestive heart failure, hepatic cirrhosis with ascites, states of aldosterone excess with normal renal function, potassium-losing nephropathy, and with certain diarrheal states

3 The use of potassium salts in patients receiving diuretics for uncomplicated essential hypertension is often unnecessary when such patients have a normal dietary pattern. Serum potassium should be checked periodically, however, and if hypokalemia occurs, dietary supplementation with potassium-containing foods may be adequate to control milder cases. In more severe cases supplementation with potassium salts may be indicated

CONTRAINDICATIONS: Potassium supplements are contraindicated in patients with hyperkalemia since a further increase in serum potassium concentration in such patients can produce cardiac arrest Hyperkalemia may complicate any of the following conditions: Chronic renal failure, systemic acidosis such as diabetic acidosis, acute dehydration, extensive tissue breakdown as in severe burns, adrenal insufficiency, or the administration of a potassium-sparing diuretic (e.g., spironolactone, triamterene).

Wax-matrix potassium chloride preparations have produced esophageal ulceration in certain cardiac patients with esophageal compression due to enlarged left atrium.

All solid dosage forms of potassium chloride supplements are contraindicated in any patient in whom there is cause for arrest or delay in tablet passage through the gastrointestinal tract In these instances, potassium supplementation should be with a liquid preparation

WARNINGS: Hyperkalemia—In patients with impaired mechanisms for excreting potassium, the administration of potassium salts can produce hyperkalemia and cardiac arrest. This occurs most commonly in patients given potassium by the intravenous route but may also occur in patients given potassium orally Potentially fatal hyperkalemia can develop rapidly and be asymptomatic. The use of potassium salts in patients with chronic renal disease, or any other condition which impairs potassium excretion, requires particularly careful monitoring of the serum potassium concentration and appropriate dosage adjustment

Interaction with Potassium Sparing Diuretics—Hypokalemia should not be treated by the concomitant administration of potassium salts and a potassium-sparing diuretic (e.g., spironolactone or triamterene) since the simultaneous administration of these agents can produce severe hyperkalemia.

Gastrointestinal Lesions—Potassium chloride tablets have produced stenotic and/or ulcerative lesions of the small bowel and deaths These lesions are caused by a high localized concentration of potassium ion in the region of a rapidly dissolving tablet, which injures the bowel wall and thereby produces obstruction, hemorrhage or perforation

K-DUR tablets contain micro-crystalloids which disperse upon disintegration of the tablet. These micro-crystalloids are formulated to provide a controlled release of potassium chloride The dispersibility of the micro-crystalloids and the controlled release of ions from them are intended to minimize the possibility of a high local concentration near the gastrointestinal mucosa and the ability of the KCl to cause stenosis or ulceration. Other means of accomplishing this (e g , incorporation of potassium chloride into a wax matrix) have reduced the frequency of such lesions to less than one per 100,000 patient years (compared to 40–50 per 100,000 patient years with enteric-coated potassium chloride) but have not eliminated them The frequency of GI lesions with K-DUR tablets is, at present, unknown K-DUR tablets should be discontinued immediately and the possibility of bowel obstruction or perforation considered if severe vomiting, abdominal pain, distention, or gastrointestinal bleeding occurs

Metabolic Acidosis—Hypokalemia in patients with metabolic acidosis should be treated with an alkalinizing potassium salt such as potassium bicarbonate, potassium citrate, potassium acetate, or potassium gluconate

PRECAUTIONS: The diagnosis of potassium depletion is ordinarily made by demonstrating hypokalemia in a patient with a clinical history suggesting some cause for potassium depletion. In interpreting the serum potassium level, the physician should bear in mind that acute alkalosis per se can produce hypokalemia in the absence of a deficit in total body potassium while acute acidosis per se can increase the serum potassium concentration into the normal range even in the presence of a reduced total body potassium. The treatment of potassium depletion, particularly in the presence of cardiac disease, renal disease, or acidosis requires careful attention to acid-base balance and appropriate monitoring of serum electrolytes, the electrocardiogram, and the clinical status of the patient

Laboratory Tests: Regular serum potassium determinations are recommended In addition, during the treatment of potassium depletion, careful attention should be paid to acid-base balance, other serum electrolyte levels, the electrocardiogram, and the clinical status of the patient, particularly in the presence of cardiac disease, renal disease, or acidosis

Drug Interactions: Potassium-sparing diuretics, see **WARNINGS.**

Carcinogenesis, Mutagenesis, Impairment of Fertility: Long-term carcinogenicity studies in animals have not been performed

Pregnancy Category C: Animal reproduction studies have not been conducted with K-DUR It is also not known whether K-DUR can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity K-DUR should be given to a pregnant woman only if clearly needed.

Nursing Mothers: The normal potassium ion content of human milk is about 13 mEq per liter. Since oral potassium becomes part of the body potassium pool, so long as body potassium is not excessive, the contribution of potassium chloride supplementation should have little or no effect on the level in human milk

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS: One of the most severe adverse effects is hyperkalemia (see **CONTRAINDICATIONS, WARNINGS,** and **OVERDOSAGE)**. There have also been reports of upper and lower gastrointestinal conditions including obstruction, bleeding, ulceration, and perforation (see **CONTRAINDICATIONS** and **WARNINGS);** other factors known to be associated with such conditions were present in many of these patients

The most common adverse reactions to oral potassium salts are nausea, vomiting, abdominal discomfort, and diarrhea These symptoms are due to irritation of the gastrointestinal tract and are best managed by taking the dose with meals or reducing the dose

Skin rash has been reported rarely

OVERDOSAGE: The administration of oral potassium salts to persons with normal excretory mechanisms for potassium rarely causes serious hyperkalemia. However, if excretory mechanisms are impaired or if potassium is administered too rapidly intravenously, potentially fatal hyperkalemia can result (see **CONTRAINDICATIONS** and **WARNINGS**) It is important to recognize that hyperkalemia is usually asymptomatic and may be manifested only by an increased serum potassium concentration and characteristic electrocardiographic changes (peaking of T-waves, loss of P-waves, depression of S-T segment, and prolongation of the QT-interval) Late manifestations include muscle-paralysis and cardiovascular collapse from cardiac arrest

Treatment measures for hyperkalemia include the following:

1 Elimination of foods and medications containing potassium and of potassium-sparing diuretics.

2 Intravenous administration of 300 to 500 ml/hr of 10% dextrose solution containing 10–20 units of insulin per 1,000 ml

3 Correction of acidosis, if present, with intravenous sodium bicarbonate

4 Use of exchange resins, hemodialysis, or peritoneal dialysis

In treating hyperkalemia, it should be recalled that in patients who have been stabilized on digitalis, too rapid a lowering of the serum potassium concentration can produce digitalis toxicity

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Auxiliary News

Alaska State Medical Association Auxiliary

The auxiliary year end is coming shortly but instead of winding down, the auxiliary is in high gear. Three projects are being developed this spring and early summer—one to earn funds for community health; another to earn money for the scholarship fund, and last but not least, our state convention.

The Auxiliary is planning the spouse program for the state convention in Sitka, and it is aimed toward including all the spouses of those attending the convention - male and female. We plan to have interesting speakers for hour-long morning sessions, and a variety of tours in the afternoons. Whenever appropriate, spouses will be invited to attend the convention meetings, lunches, and dinners with the physicians.

A garage sale will be held in May with the proceeds going to the Alaska Lung Association for their Champ Camp for young asthmatics. A series of dinners will be held in early summer to raise money for the state scholarship fund which gives one or two $1000 scholarships to Alaskans studying health care.

Hope to see you all in Sitka this June.

Alice Samuelson
President, ASMA Auxiliary

Anchorage Medical Society Auxiliary

It has been a very busy year for the Anchorage Auxiliary, starting with politics. In September we held a political action workshop followed by luncheon with the candidates and incumbents. Forty participants listened to Senator Arliss Sturgulewski, Representative Johnny Ellis, and Speaker of the House, Sam Cotten tell us how to become more effective in our efforts to impact the legislature and how to use this information in our Proposition 2 efforts.

As part of our commitment to Proposition 2 and meaningful tort reform, the auxiliary provided support people in the Citizen's Coalition for Tort Reform office and helped organize the fund-raising auction, which helped to pay for the ad campaign. Our volunteers distributed brochures to physician offices in the community. We comprised one half of the volunteer force who distributed 2,500 brochures in a door-to-door neighborhood walk just before the election.

Thanks to the efforts of many people Proposition 2 passed by a large margin. We haven't sat back and rested on our laurels, though. We still worked to be sure that the bill is passed this session.

Politics was not our only interest, however. The fall also brought our annual health projects fund raiser, a masked dance. Proceeds totalling in excess of $2,000.00 covered the cost of Children's Healthfest '89 held earlier this month.

Children's Healthfest was the largest project of the year. It required a lot of time, effort, organization, and was truly the gift to our community it was meant to be. Once again our fair was well organized, well attended, and well received by the community—due in no small part to the efforts of hard working auxiliary volunteers. Many thanks to all the physicians who spent part of their day off examining children (and a few parents) in the aisles of Northway Mall.

The auxiliary is also aware of the special health needs of adolescents and were happy to help with clothing, linen, and book donations throughout the year to newly opened Covenant House for runaway teens. Guests at the Holiday Tea were asked to bring new hats and mittens for mothers and children staying at Claire House. General membership meetings focused on volunteer opportunities in the community, and more recently, Dr. Patterson introduced us to the impaired physician program. The auxiliary is currently establishing a support committee to work with the impaired physician committee.

Though spring is here, signalling a less active time for the auxiliary, we still have a few things planned for you. We hope you attended the Spring Luncheon and Fashion Show on May 17, 1989, at O'Malleys on the Green.

My year as president has come to a close. I would like to express my thanks to all those people who worked on the various projects sponsored by our organization. Any project, large or small, requires the effort of many people. Those people who are highly visible readily receive our thanks, but those working behind the scenes don't always get the recognition they deserve. To those people, who gave of themselves and their time, thank you.

Thank you all for the opportunity to work with you and for your encouragement and support this year.

Lindsay Hanson Tucker
President, 1988-1989

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Editorial

I tried mightily to kindle a spark of inspiration so this issue's editorial would be on a subject other than the oil spill. I failed. Things looked good for several days, but then the spark was extinguished by recent news that under regulations recently finalized by the Department of Labor, Occupational Safety and Health Administration (OSHA), petroleum spills are considered as hazardous wastes. As a result, all workers involved in clean-up activities are covered by stringent requirements that mandate employers to provide at least 40 hours of training; to establish medical monitoring, short- and long-term surveillance; and to keep all medical and surveillance records for at least 30 years.

* * * * * * *

Plans now are being made to obtain medical and toxicologic consultation from unions, industry, and federal and state agencies to assess potential health effects from exposure to Alaskan crude oil during clean-up activities.

* * * * * * *

Major efforts of public health agencies until now have concentrated on insuring the physical safety of those involved in clean-up activities. The Alaska Department of Conservation (DEC), as if it didn't already have enough problems to deal with, has had responsibility for insuring safe water, safe food preparation and restaurant inspections, and safe solid waste disposal for the thousands drawn to Valdez and surrounding areas. The Alaska Division of Public Health has had its Emergency Medical Services professionals in Valdez. It is a tribute to all that there have been no deaths or serious injuries, considering the severe physical environment, long hours, and dangerous work of so many.

* * * * * * *

There has been a great deal of public discussion about the role of industry in the spill and clean-up, but surprisingly little meaningful discussion about the role of alcohol—discussion that is not just bashing the captain and crew for what happened that fateful night. How have our society's values and structures evolved into a system that allowed a company like Exxon to put a $125 million tanker under the command of an individual with an apparently well-documented, severe drinking problem?

* * * * * * *

- Why did the captain's employers not intervene at some time in the past? After all, within 48 hours of the wreck, newspapers had reported the captain's record of three past episodes of driving while intoxicated, with the last conviction in September 1988.

- We routinely order many tests. We should begin to obtain blood alcohol levels on all our patients who are injured and seek medical care. Like any problem, one documented early may lead to more effective intervention.

* * * * * * *

There are 231 cubic inches in one gallon. If the spill released 11 million gallons, the oil would cover an area of 0.63 square miles one inch deep.

* * * * * * *

There are many questions that have no reply. In the months to come, I hope we will examine the causes of the Alaskan oil spill. One thing is for certain. The spill was *not* a random and unpredictable event. President Bush was dead wrong when he called the spill an "accident."

* * * * * * *

1989 marks the 30th anniversary of *Alaska Medicine.* Founded by William J. Mills, M.D., in 1959, the journal has been an important voice to enhance science, medicine, and public health in Alaska. Many of our highly esteemed colleagues have shared in the responsibility of producing *Alaska Medicine*. Many have contributed articles. The journal exists as living testimony to Alaska's physicians and their commitment to excellence, integrity, and the highest standards of medicine.

* * * * * * *

Alaska Medicine is beginning year three of its five year growth and development plan. We hope you, our readers, are pleased with our journal and its content. Many changes have been made, and more lie ahead.

Alaska Medicine could not exist without the commitment of Alaska's physicians and the support of the Alaska State Medical Association.

* * * * * * *

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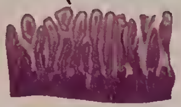
Aspirin and other nonsteroidal anti-inflammatory drugs weaken mucosal defenses, which may lead NSAID users to become prone to duodenal ulcers![1] For those NSAID users who do develop duodenal ulcers, CARAFATE® (sucralfate/Marion) is ideal first-line therapy. Carafate rebuilds mucosal  defenses through a unique, nonsystemic mode of action. Carafate enhances the body's natural healing ability while it protects damaged mucosa from further injury. So the next time you see an arthritis patient with a duodenal ulcer, prescribe nonsystemic Carafate: therapy for the ulcer-prone patient.

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Carcinogenesis, Mutagenesis, Impairment of Fertility: Chronic oral toxicity studies of 24 months' duration were conducted in mice and rats at doses up to 1 gm/kg (12 times the human dose). There was no evidence of drug-related tumorigenicity. A reproduction study in rats at doses up to 38 times the human dose did not reveal any indication of fertility impairment. Mutagenicity studies were not conducted.

Pregnancy: Teratogenic effects. Pregnancy Category B. Teratogenicity studies have been performed in mice, rats, and rabbits at doses up to 50 times the human dose and have revealed no evidence of harm to the fetus due to sucralfate. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when sucralfate is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS

Adverse reactions to sucralfate in clinical trials were minor and only rarely led to discontinuation of the drug. In studies involving over 2,500 patients treated with sucralfate, adverse effects were reported in 121 (4.7%).

Constipation was the most frequent complaint (2.2%). Other adverse effects, reported in no more than one of every 350 patients, were diarrhea, nausea, gastric discomfort, indigestion, dry mouth, rash, pruritus, back pain, dizziness, sleepiness, and vertigo.

OVERDOSAGE

There is no experience in humans with overdosage. Acute oral toxicity studies in animals, however, using doses up to 12 gm/kg body weight, could not find a lethal dose. Risks associated with overdosage should, therefore, be minimal.

DOSAGE AND ADMINISTRATION

The recommended adult oral dosage for duodenal ulcer is 1 gm four times a day on an empty stomach.

Antacids may be prescribed as needed for relief of pain but should not be taken within one-half hour before or after sucralfate.

While healing with sucralfate may occur during the first week or two, treatment should be continued for 4 to 8 weeks unless healing has been demonstrated by x-ray or endoscopic examination.

HOW SUPPLIED

CARAFATE (sucralfate) 1-gm tablets are supplied in bottles of 100 (NDC 0088-1712-47) and in Unit Dose Identification Paks of 100 (NDC 0088-1712-49). Light pink scored oblong tablets are embossed with CARAFATE on one side and 1712 bracketed by C's on the other. Issued 1/87

Reference:

1. Eliakim R, Ophir M, Rachmilewitz D: *J Clin Gastroenterol* 1987;9(4):395-399.

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Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

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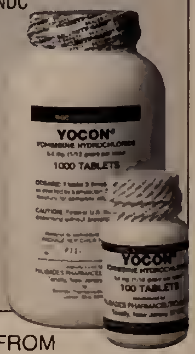

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1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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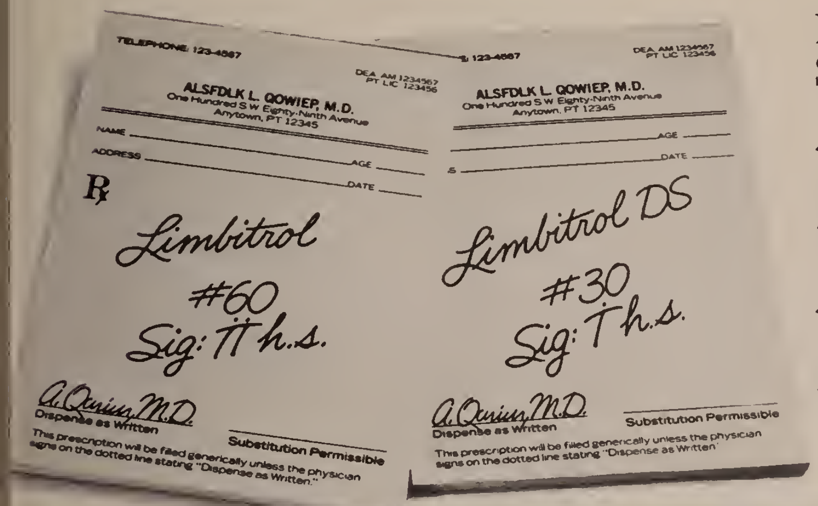

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References: 1. Data on file, Hoffmann-La Roche Inc., Nutley, NJ. 2. Feighner JP, *et al: Psychopharmacology 61:*217-225, Mar 22, 1979.

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Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants; concomitant use with MAOIs or within 14 days of monoamine oxidase inhibitors (then initiate cautiously, gradually increasing dosage until optimal response is achieved); during acute recovery phase following myocardial infarction.

Warnings: Use with caution in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur when used with anticholinergics. Closely supervise cardiovascular patients. Arrhythmias, sinus tachycardia, prolongation of conduction time, myocardial infarction and stroke reported with tricyclic antidepressants, especially in high doses. Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (*e.g.,* operating machinery, driving).

***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations. Consider possibility of pregnancy when instituting therapy.**

Withdrawal symptoms of the barbiturate type have occurred after discontinuation of benzodiazepines (see Drug Abuse and Dependence).

Precautions: Use cautiously in patients with a history of seizures, in hyperthyroid patients, those on thyroid medication, patients with impaired renal or hepatic function. Because of suicidal ideation in depressed patients, do not permit easy access to large quantities of drug. Periodic liver function tests and blood counts recommended during prolonged treatment. Amitriptyline may block action of guanethidine or similar antihypertensives. When tricyclic antidepressants are used concomitantly with cimetidine (Tagamet), clinically significant effects have been reported involving delayed elimination and increasing steady-state concentrations of the tricyclic drugs. Use of Limbitrol with other psychotropic drugs has not been evaluated; sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Should not be taken during the nursing period or by children under 12. In elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects. Inform patients to consult physician before increasing dose or abruptly discontinuing this drug.

Adverse Reactions: Most frequent: drowsiness, dry mouth, constipation, blurred vision, dizziness, bloating. Less frequent: vivid dreams, impotence, tremor, confusion, nasal congestion. Rare: granulocytopenia, jaundice, hepatic dysfunction. Others: many symptoms associated with depression including anorexia, fatigue, weakness, restlessness, lethargy.

Adverse reactions not reported with Limbitrol but reported with one or both components or closely related drugs: *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke. *Psychiatric:* Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania, increased or decreased libido. *Neurologic:* Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns. *Anticholinergic:* Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue. *Endocrine:* Testicular swelling, gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female, elevation and lowering of blood sugar levels, and syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Other:* Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.

Drug Abuse and Dependence: Withdrawal symptoms similar to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of chlordiazepoxide; more severe seen after excessive doses over extended periods; milder after taking continuously at therapeutic levels for several months. Withdrawal symptoms also reported with abrupt amitriptyline discontinuation. Therefore, after extended therapy, avoid abrupt discontinuation and taper dosage. Carefully supervise addiction-prone individuals because of predisposition to habituation and dependence.

Overdosage: Immediately hospitalize patient. Treat symptomatically and supportively. I.V. administration of 1 to 3 mg physostigmine salicylate may reverse symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.

How Supplied: *Double strength (DS) Tablets,* white, film-coated, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt), and *Tablets,* blue, film-coated, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 50.

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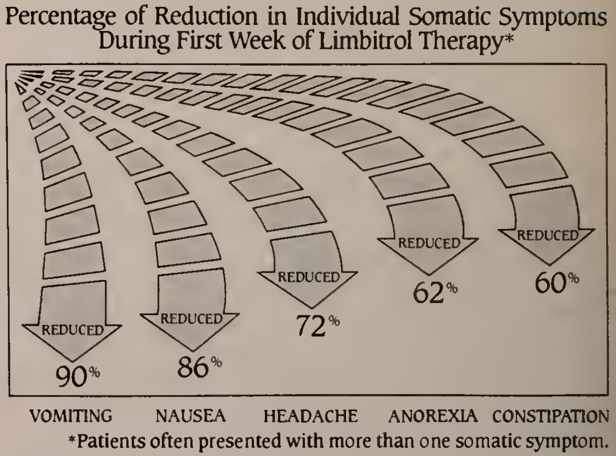

*Patients often presented with more than one somatic symptom.

Caution patients about the combined effects of Limbitrol with alcohol or other CNS depressants and about activities requiring complete mental alertness, such as operating machinery or driving a car. In general, limit dosage to the lowest effective amount in elderly patients.

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Renovascular Hypertention and Demyelimating Disease in a Young Woman

by Richard L. Neubauer, M.D.[1]

CASE REPORT

History

A 28-year old woman was admitted to the hospital with severe hypertension and transverse myelitis in March, 1984.

She had a history of tingling in her toes for one and a half months which had begun to ascend to her waist for one week prior to her admission. She had also started to stumble, because she was unable to sense the position of her lower extremities.

In addition, she had started having severe retro-orbital headaches for a few months, which would fluctuate from one side of her head to the other, and which were made much worse by alcoholic beverages. Seven months earlier, her blood pressure had been measured and was "borderline." One week earlier, her blood pressure had been measured at a clinic on the North Slope oil fields in Prudhoe Bay where she worked as a heavy equipment operator, and had been 190/100.

Past medical history was significant in that she was taking no medications. Four years previously she had an episode of bilateral optic neuritis, diagnosed at another hospital, and treated with a course of prednisone. She had minimal residual visual loss, although she described "near total blindness" at the time of the illness.

Physical Examination

Blood pressure was 214/128 in the left arm, and 196/116 in the right. She was in no acute distress but was uncomfortable from her leg symptoms. Funduscopic examination showed marked arteriolar narrowing and evidence of optic atrophy. Examination of the heart and lungs was normal. A faint bruit was heard on the left side of her abdomen.

Neurologic examination showed decreased vibratory sense in both legs, the left worse than the right. Pinprick sensation was normal, but light touch sensation was felt poorly to the T7 level. In addition there was a band of dysesthesia to pinprick at the T7 level. Motor examination, and reflexes were normal and both Babinskis were downgoing.

Laboratory Values

Electrolytes included a sodium of 140 mEq/L, potassium 3.4 mEq/L, chloride 102 mEq/L, and bicarbonate was 33 mEq/L. A urinalysis showed trace protein and no cellular elements. EKG and chest x-ray were normal. Plasma renin, done while the patient was supine and on a normal diet, was 3.1 ng/ml (nl 0-2.1 ng/ml). Urine metanephrines were 0.3 mg/L and urine VMA was 4.6 mg/l, both normal. CBC was normal. Computed tomography of the abdomen did not show any evidence of adrenal masses.

A lumbar, thoracic, cervical myelogram was done to exclude a structural cause of the myelopathy, and was normal. An RPR was negative and serum B12 value was normal. Both ANA and Rheumatoid Factor were negative. Spinal fluid showed a normal value for glucose and protein, and a test for oligoclonal bands was negative. Brainstem evoked response testing was interpreted as consistent with a demyelinative process, central and bilateral. Posterior tibial somatosensory evoked responses showed a lack of transmission of sensory input through the posterior aspect of the spinal column consistent with a demyelinative disease.

Subsequent Course

The patient was treated with prednisone and had a gradual resolution of the symptoms in her lower extremities over a few weeks. She was started on atenolol and initially her blood pressure fell to 130/80, and she was discharged from the hospital.

One month later, she had recurrence of headaches, and her blood pressure was again 172/110. A digital subtraction renal angiogram was done via arterial injection, and showed an area of marked narrowing in the left renal artery immediately proximal to its bifurcation, with beading distal to that point (Figure 1). The procedure was complicated by a vasovagal episode at the time of catheter manipulation that responded to atropine. Split renal vein renin determinations were not done.

She then underwent successful balloon angioplasty of the lesion, after pretreatment with captopril and hydration (Figure 2 and 3). She was discharged from the hospital normotensive, and has been followed off

---

[1] 2841 DeBarr Road, Suite 23, Anchorage, Alaska 99508

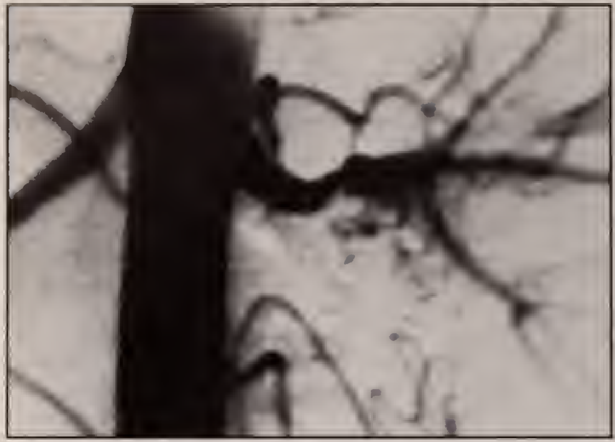

Figure 1. Digital Subtraction angiogram via arterial injection showing "beaded" left renal artery and collateral vessels. (Photograph courtesy of Richard Hill M.D.)

all medications for five years during which time she has remained normotensive.

She has had several minor episodes of numbness primarily in the lower extremities, which were interpreted as possible episodes of demyelinative disease, and which were treated with brief courses of corticosteroids. These episodes have all been self limited. A magnetic resonance imaging scan was done when that became available, and this showed two small areas of T2 signal in the centrum semiovale posteriorly on the left and anteriorly on the right consistent with small focal areas of demyelination. A second MRI scan two years later showed progression of the areas of demyelination in the periventricular white matter consistent with multiple sclerosis.

DISCUSSION

Etiology and Pathogenesis of Renovascular Hypertension

Renovascular hypertension may occur as a result of a variety of vascular and extravascular processes. Approximately two thirds of cases are related to unilateral or bilateral atherosclerotic narrowing of the renal arterial system. Most of the remaining cases are related to fibromuscular dysplasia of the renal vessels. A small minority of cases may be caused by other rare vascular diseases including congenital arteriovenous malformations or fistulas, congenital coarctation of the abdominal aorta, neurofibromatosis, Takayasu's aortitis, thrombosis or embolization of the renal artery, or radiation vasculitis. Extravascular compression of the renal arterial system may also lead to the syndrome. Renovascular hypertension may occur even in a pediatric population (1).

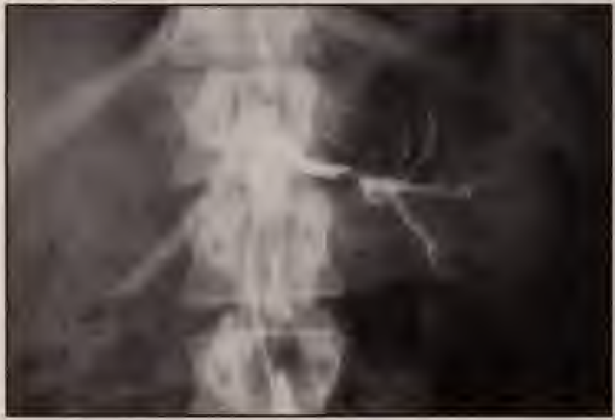

Figure 2. Selective injection showing stenotic area during angioplasty. Photograph courtesy of Richard Hill M.D.)

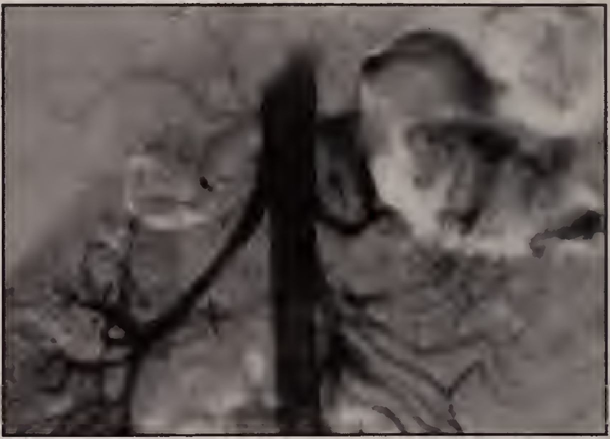

Figure3. Post dilatation view showing successful expansion of the left renal artery stenosis (Photograph courtesy of Richard Hill, M.D.)

While as few as 1% of all hypertensive patients may have a specific, curable reason for their elevated blood pressure, renovascular disease is probably the most common etiology found in this group. Furthermore, in patients with severe or accelerated hypertension, a much larger percentage of patients may be found to have a renovascular cause for their illness.

The anatomy and classification of renovascular lesions may be important in deciding on a rational treatment plan. In atherosclerotic disease, renal artery stenosis may be the only demonstrable manifestation, or may occur in conjunction with generalized vascular disease. Atherosclerotic lesions may be located at the ostium to the renal artery in association with aortic plaques, in the main renal artery, and/or in branch vessels (2).

Fibromuscular dysplasia may be classified according to the layer of the artery that is affected (3). A unifying classification (table 1) has been developed to describe these lesions (4). Medial fibroplasia is most common and appears as a multifocal "string of beads." These usually start in the mid portion of the renal artery. Perimedial fibroplasia is the second most common morphology, and often results in a severely stenotic lesion with smaller "beads". Intimal fibroplasia is usually seen as a sharply demarcated, often profound, stenosis which may be found in all age groups and is felt to possibly represent a local response to injury. Medial hyperplasia is produced by arterial smooth muscle hyperplasia, and appears as a concentric segmented stenosis. Periarterial fibroplasia is uncommon and appears as a segmental tubular stenosis.

The cause of these lesions remains unknown. Women are affected more often than men. Fibromuscular dysplasia affects a significantly younger age group that does atherosclerotic disease. The onset of hypertension before age 30 is a clinical clue to the possible presence of renovascular hypertension.

Whatever the underlying cause of the renal artery stenosis, hypertension in this condition is felt to be mediated at least in part by the renin-angiotensin-aldosterone system. Diminished renal perfusion distal to the stenosis results in a release of renin which in turn increases plasma angiotensin II levels, which in turn causes vasoconstriction. In addition, angiotensin stimulates aldosterone secretion which causes sodium retention and volume expansion which may also contribute to hypertension.

Over time, as the hypertension continues, other mechanisms may come into play that sustain an elevated blood pressure. Damage to the contralateral kidney may, for instance, become a factor. Especially in an older age group, essential hypertension may coexist with renovascular hypertension. Evaluating the extent to which a stenotic renal vessel contributes to hypertension may sometimes be difficult.

Table 1
Classification of Fibromuscular Dysplasia

Intimal fibroplasia	lesions may be hard to dilate
Medial fibroplasia	60-80% of lesions "string of beads"
Medial hyperplasia	concentric stenosis
Perimedial fibroplasia	10-25% of lesions smaller "beads"
Periarterial fibroplasia	least common

Clinical Features and Diagnosis

Clinical clues to the presence of renovascular hypertension are outlined in Table 2. A high index of suspicion is required, because there are no pathognomonic findings that point unequivocally to the diagnosis (5). In the case presented, there were several clues to the presence of renovascular hypertension, including age less than 30, recent onset of fairly severe hypertension, the presence of an abdominal bruit, mild proteinuria, and hypokalemia.

Several items from Table 2 are worthy of special consideration. Reversible renal failure related to the use of angiotensin converting enzyme (ACE) inhibitors has been reported in patients with bilateral renal artery stenosis or renal artery stenosis in a solitary kidney. The mechanism by which this occurs is felt to be related to disruption of the autoregulation of glomerular filtration rate (GFR) by interference with the renin-angiotensin system (6). Specifically, it is felt that this is related to a combination of diminished renal arterial perfusion pressure combined with loss of efferent (postglomerular) arteriolar tone directly related to pharmacologic blockade of the renin-angiotensin system (7,8). Proteinuria has recently been observed in association with renal artery stenosis, and has been found to diminish with successful treatment of the stenosis using renal angioplasty (9).

Table 2
Clinical Clues to Renovascular Hypertension

History
1) Age under 30 or recent onset of HTN after age 50
2) Accelerated or malignant HTN
3) HTN refractory to a 3 drug medical regimen
4) Reversible azotemia assoc. with ACE inhibitors
5) Unilateral small kidney by any study

Physical Examination
1) Severe retinopathy
2) Continuous abdominal or flank bruit
3) Other evidence of atherosclerotic disease
4) related disease (eg. neurofibromatosis)

Laboratory
1) Hypokalemia (related to hyperaldosteronism)
2) Azotemia
3) Proteinuria (>150 mg/24 hours)

A variety of screening studies to detect renovascular lesions in a population of hypertensive patients have been evaluated, but none has been found to be widely acceptable. Rapid sequence intravenous pyelography is reasonably sensitive, detecting up to 80% of cases, but lacks specificity. Plasma renin activity is felt to be a poor screening test, primarily because it must be indexed to dietary sodium intake, the position of the patient during the test, and to drugs that the patient may be taking. Also, a high renin value may be present in a substantial percentage of patients with essential hypertension.

A stimulation test, using angiotensin converting enzyme inhibitors has been devised, and may have some benefit in evaluating patients who may be at high risk for renovascular lesions on clinical grounds. The test exploits a hyper-reninemic response to ACE inhibition in patients with renovascular hypertension versus other forms of hypertension. This test may be performed by administering 25 mg of captopril to a seated patient on a normal diet. Baseline plasma renin activity (PRA) and blood pressure are compared at baseline and 60-90 minutes. A blood pressure decrease of 10% and PRA increase of 2.5 times the control (or to levels > 10 ng/ml/hr) are felt to be most predictive (10). This test has the disadvantage that to be most easily and reliably interpreted, the patient must be off anti-hypertensive medications. Its sensitivity and specificity have also not yet been determined.

Isotope renography has been found to have an unacceptably low specificity in detecting renovascular hypertension. However, isotope renography performed with administration of captopril shows some promise in identifying patients with renovascular disease in appropriately selected high risk patients (11). Renography with captopril takes advantage of the functional decrease in GFR associated with ACE inhibitors in the kidney affected by an arterial stenosis. This decreased GFR in the kidney affected by the stenotic lesion (in comparison with the other "normal" kidney) is detected with the isotope renogram by assessing time-activity curves or uptake as an estimate of GFR. While the concept holds promise, the role of this study in evaluating patients with suspected renovasular hypertension is not yet fully determined.

Angiography is the "gold standard" for detecting and characterizing renal artery stenosis once it is suspected. Digital subtraction angiography (DSA) via venous injection has the advantage of not requiring an arterial puncture, but potentially may require more contrast material than standard angiography. There are additional disadvantages to venous DSA in evaluating renal artery stenosis (12). This technique may underestimate the extent of stenosis with atheromatous lesions, albeit usually only to a minor extent. However, in fibromuscular lesions, the degree of stenosis may be greatly underestimated particularly if the lesion is very short. For these reasons, venous DSA cannot be recommended in the evaluation of renal artery stenosis. Arterial DSA may approach conventional angiography in diagnostic value, and has the advantage of utilizing lower doses of contrast material. It must be remembered that while angiography detects the stenotic lesion, it does not assess the functional relationship between the stenosis and the elevated blood pressure.

Split renal vein renin values may be of help in deciding the functional significance of a lesion. A ratio of 1.5:1 between the renal veins is deemed significant, and may be of predictive value in deciding whether or not to consider interventional therapy. However, the utility of these determinations may be limited if bilateral renal artery disease is present and a variety of technical factors make the results of such determinations difficult to interpret at times. A high false negative rate has been reported, in that many patients with non-lateralizing values may obtain a beneficial result if intervention is undertaken anyway.

Treatment

There are three treatment options for renovascular hypertension: pharmacotherapy, renal angioplasty, or reconstructive vascular surgery to relieve the stenosis. Nephrectomy, once the only option for surgical treatment of renal artery stenosis, is now rarely performed.

Pharmacotherapy may be an option in certain cases, especially in an older patient with renovascular hypertension of atherosclerotic origin. The trend does appear to be toward "interventional" therapy with either angioplasty or surgery, but patient preference, or other complicating factors may lead to attempts at controlling blood pressure with medications.

Since their introduction, ACE inhibitors have taken on an important role. While they block the conversion of angiotensin-I to angiotensin-II, they also have a more complex mechanism of action which ultimately results in decreased peripheral resistance without a change in cardiac output or in heart rate. They may be effective in a hypertensive patient regardless of plasma renin activity. As has already been mentioned, care must be exercised in the use of these agents because of the (reversible) azotemia that has been observed in patients with renal artery stenosis.

Beta adrenergic blocking agents have also demonstrated particular efficacy in patients with renal artery stenosis, and it appears that these drugs are more effective in patients with elevated plasma renin activity.

In general, if pharmacotherapy is chosen, care should be taken to monitor renal function since loss of function may occur due to decreased renal perfusion as the blood pressure is lowered.

Percutaneous transluminal angioplasty (renal angioplasty) has rapidly ascended to an important place in the management of these patients. The technique is limited mainly by anatomic considerations. For instance, lesions at the ostium of the renal artery may be technically more difficult and the complication rate may be higher in dilating lesions at this location. The experience of the team performing the procedure appears to play a role in success and complication rates. Currently, it seems that the limits of this procedure are still being explored, and in certain patients the technique is even being applied to totally occluded renal arteries in an effort to salvage renal function.

Success rates with angioplasty vary with the type of lesion. Fibromuscular dysplasia responds best to the technique, and may yield long term improvement in up to 75% of patients (13). One type of fibromuscular dysplasia, the intimal type, may be harder to dilate. The lesions of Takayasu's aortitis and neurofibromatosis may also be difficult to dilate. Additional problems may be encountered in crossing long lesions with the guide wire. Some lesions may be multiple, and stenoses in branch vessels cannot be reached with current techniques. Thus, the length and location of the lesions are often key to success or failure (14). Pretreatment of the patient with ACE inhibitors before angioplasty may prevent a precipitous drop in blood pressure after dilation (15).

Angioplasty of atheromatous lesions is generally more technically difficult (16). As with fibromuscular dysplasia, short lesions tend to be most amenable to dilatation. Ostial lesions pose special difficulties and the risk of embolization is of particular concern. Angioplasty of totally occluded vessels may be attempted if the size and residual function of the kidney warrants salvage, and if the patient's clinical condition and the appearance of the lesion are favorable.

Indications for angioplasty of atheromatous lesions are usually either refractory hypertension or hypertension associated with deteriorating renal function; angioplasty may benefit both (17,18). The overall long term success rate in terms of improved blood pressure control and/or improved renal function are less than in fibromuscular dysplasia, but about a third of patients obtain longstanding benefit.

Surgical therapy for renal artery stenosis has also evolved in recent years (19). Long term success rates may be higher than with angioplasty, especially in elderly patients (20,21). Improved long term survival also appears to be demonstrable, even in patients with generalized atherosclerosis (22). Newer techniques include ex vivo reconstruction of vascular lesions that may not be accessible to standard surgical techniques (23).

Complications of Angioplasty

Complications of angioplasty can be categorized as those associated with the puncture site, those at the site of the dilatation, and distant and generalized complications (24). The overall complication rate is 10-20% depending on the experience of the operator (25). Complications requiring surgery occur in 2-5% of patients, and it has been recommended that an experienced vascular surgeon be readily available at the time of angioplasty. Renal failure may occur related to contrast agent. The risk of renal failure depends on the patient's risk factors for contrast agent toxicity including age, presence of diabetes, baseline renal function, etc..

Dissection or thrombosis of the main renal artery or its branches may occur, usually secondary to trauma while crossing the stenotic lesion. Embolization is rare, but the consequences may be serious. Usually this occurs in patients with extensive atherosclerosis. Rupture of the renal artery may occur, and may even be delayed for days after angioplasty due to severe weakening of the vascular wall (26,27).

Choosing a therapeutic approach depends on careful consideration of the age of the patient, the status of renal function, the overall clinical status of the patient, and the responsiveness of the blood pressure to pharmacotherapy. Controlled studies comparing the therapeutic options are not available (28,29).

Are this Patient's Neurologic Symptoms Related to Fibromuscular Dysplasia?

The neurologic symptoms in the case include a remote, but well documented episode of optic neuritis, transverse myelitis, and a series of ill-defined episodes of patchy sensory findings in the last few years. There have also been findings demonstrated in somatosensory evoked responses, and brainstem evoked responses that are compatible with a demyelinating process, and she has been presumed to have multiple selerosis. Could these symptoms, however, be secondary to fibromuscular dysplasia in the cerebral, and spinal vasculature?

Fibromuscular dysplasia is an angiopathy of unknown etiology which was first described in association with the renal vasculature. However, it has since been recognized as, at least in some cases, a systemic disease. The subcategories of fibromuscular dysplasia have already been discussed. These entities usually affect medium sized arteries, and may affect the carotid and vertebral arteries, in addition to the renal arteries. In some cases, the condition may be familial (30).

Fibromuscular dysplasia of the cephalic circulation has been reported with or without concomitant disease of the renal vasculature (31). The presence of fibromuscular dysplasia may be associated with intracranial aneurysm, embolic phenomena, or dissection of the affected vessels (32). Clinical symptoms include focal neurologic symptoms (stroke, amaurosis fugax, transient ischemic attacks), subarachnoid hemorrage from a ruptured intracranial aneurysm, or symptoms of global ischemia (33).

In the case reported, it is unlikely that the spectrum of symptoms, physical findings, and history could be related to fibromuscular dysplasia. Specifically, the transverse myelitis would be difficult to explain on this basis. Involvement of the spinal arteries would have to be hypothesized, and I could find no support in the medical literature for past reports of such involvement.

SUMMARY

Renal artery stenosis is an uncommon, but curable cause of systemic hypertension. The most common causes of stenosis are atherosclerosis, and fibromuscular dysplasia. Diagnosis may be difficult due to the lack of a suitable screening test, and a high index of suspicion needs to be maintained. Treatment may be with pharmacotherapy, renal angioplasty, or surgery and the choice of therapy needs to be tailored to the individual patient. Once a stenotic lesion is discovered in a hypertensive patient, the functional significance is not always clear cut.

Fibromuscular dysplasia may be a systemic disease in some cases, and may affect the central nervous system.

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Sudden Unexpected Death In Young Athletes: A Review

by Ronald E. Christensen, M.D.[1]

ABSTRACT

Sudden death is a startling and tragic event that often sends ripples through our communities and even across our nation. Estimates have suggested that between 10 and 30% of adults die suddenly and unexpectedly and maybe 5% of these are related to sports (1). The purpose of this study is to examine the topic of sudden death in the young athlete through a review of several larger national studies. Then the etiology of these tragedies will be discussed. Finally, a review of the sudden death in adolescents and young adults in Alaska over the last 12 months will be presented.

INTRODUCTION

The first recorded sudden death of an athlete was documented in about 490 B.C. when Pheidippides raced 26 miles to Athens to announce the retreat of the Persians across the Plain of Marathon. He is said to have exclaimed "Rejoice, we conquer," then suddenly fell dead. This, of course, gives rise to our annual torch race of the modern Olympic games.

Pete Maravich collapsed and died of a cardiac arrest at age 40, during a pick-up basketball game on January 5, 1988. This death shocked the public and surprised the experts. Not only was he relatively young, but also had absence of risk factors for atherosclerosis.

Post mortem examination revealed "cardiomyopathy due to chronic ischemic myocardial fibrosis due to anomalous single right coronary artery."(2) He did not have a left coronary artery. He had only minimal atherosclerosis. Extensive myocardial fibrosis showed that the ischemic condition was chronic and generalized.

Flo Hyman, an Olympic volleyball player, died suddenly in January, 1986 during a nationally televised volleyball tournament. Her autopsy showed that she had Marfan's Syndrome and a ruptured aortic aneurysm. This reveals that even our Olympic volleyball players are at risk.

Could these cardiovascular time bombs have been predicted or even detected? Maravich's only apparent symptoms were chest and shoulder discomfort, not unusual musculoskeletal complaints for an athlete. These deaths continue to raise questions of awareness, detection and prevention of sudden death.

EXERCISE AND SUDDEN DEATH

It has been argued that exercise does not necessarily have a causal link to sudden death. Koplan has estimated about 100 deaths per year during jogging alone, based on chance. Fortunately the incidence of premature death in athletes is low especially when considering the large number of competitors;

- 1/396,000 persons hours of jogging,
- 1/13,000 to 26,000 person hours of cross country skiing,
- 1/50,000 player hours of rugby (3).

The other side of the argument is reflected in a review of Rhode Island deaths (4) that suggested the risk of sudden death from coronary artery disease while jogging was seven times the rate of sudden death during sedentary activity. Cross country skiers in Finland had four times the rate of sudden death as that of individuals at rest (5). Gibbons and colleagues studied almost 3,000 adults over a 65 month period (6). These individuals had resting and maximal exercise EKGs performed and were required to keep computer logs of their activity. They entered a total of 375,000 hours of exercise. Two nonfatal cardiac events occurred during this period. The result of this study showed that maximal risk for cardiac events during exercise ranged from 0.45 to 4.3 events per 10,000 person hours depending on intensity of exercise.

In other words, data exits to argue that exercise can hasten one's demise as well as improve his outlook. This dilemma is resolved when one realizes that certain individuals with predisposing factors are at higher risk of sudden death.

PREDISPOSING FACTORS - NON CARDIAC

What conditions have been identified as predisposing factors for premature and sudden death? It is useful from an anatomic and clinical point of view to categorize sudden death victims into those dying over age 30 and those dying under age 30.

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In a recent review (7) virtually all sudden deaths in individuals older than 30 were related to coronary artery disease.

In those under 30 years of age the issues become more complex. In this group sudden death can be classified as cardiac or non-cardiac. Most of these deaths can be attributed to cardiac lesions, but coronary artery disease, that is atherosclerosis, plays a minor role.

Asthma, intracranial hemorrhage and infections all act to increase one's risk of sudden death. Some argument occurs when one looks at sickle cell trait as a predisposing factor. Kark and his coworkers (8) studied sudden death among U.S. Armed Forces recruits and found a 28 to 40 times greater risk in those with sickle cell trait than those without. Eichner (9) proposes that it might not be the trait but related renal abnormalities, the sudden load of myoglobin and potassium from the severe muscle breakdown, that causes increasing risk of fatal arrhythmia.

CAUSE OF DEATH - NON CARDIAC

In one article, Neuspiel, et al. (15) reviewed the cases of 207 sudden and unexpected non traumatic deaths. Infection, including bronchopneumonia, septic shock, meningitis and epiglottitis made up the largest group (N=59) of sudden death.

The second largest group were those with cardiac lesions (N=51).

Of the 20 deaths from intracranial hemorrhage, the most common etiologies were vascular malformations and aneurysms (N=10). These are often hereditary.

Asthma was the terminal event in 11 of the 207 victims and apparently was previously documented. The role of therapy in these deaths is unknown.

PREDISPOSING FACTORS - CARDIAC

Cardiovascular factors in sudden death can be further categorized into structural and conductive abnormalities. Structural abnormalities include: 1.) coronary artery anomalies, 2.) myocardial, 3.) valvular and 4.) aortic factors. Among the conductive factors are: 1.) AV node dysfunction such as abnormal AV nodal bundles and conductive pathways, 2.) the idiopathic long QT interval syndrome, and 3.) other abnormalities.

PREDISPOSING FACTORS - CORONARY ARTERY ANOMALIES

In his review of 147 athletes who were victims of sudden death, Waller (10) observed that congenital coronary artery anomalies were the most common predisposing factors. None had previous clinical symptoms of cardiovascular disease and all died during or shortly after vigorous physical activity. The most common anomaly was the origin of the left coronary artery from the right sinus of Valsalva. It is believed that the passage of the artery between the aorta and pulmonary trunk causes an acute angle take-off with a compromised or slit-like opening. This may kink the coronary artery during systole and may be compounded during exercise. The anomaly that Peter Maravich died from is extremely rare. He had a single right coronary artery but no left coronary artery, combined with marked cardiac hypertrophy.

Exercise-induced coronary artery spasm has been proposed as a cause of death in some athletes. One report described 13 subjects with exercise-induced coronary spasm, all of whom had abnormal ST segment elevation during exercise but only two of whom had afternoon exercise EKG changes (Yasue) (11). Afternoon exercise might be safer in those individuals known to have coronary artery spasm.

PREDISPOSING FACTORS - MYOCARDIAL

Abnormalities in the muscle of the heart are frequently found disorders in young victims of exercise-related sudden death. In his review of 29 athletes Maron et al.(12), found that hypertrophic cardiomyopathy was the most commonly identified cardiovascular lesion (only three had coronary artery anomalies, 14 had hypertrophic cardiomyopathy).

Hypertrophic cardiomyopathy (H.C.M.) is a condition defined as a hypertrophied non-dilated left ventricle with asymmetric septal hypertrophy, and marked disorganization of cardiac muscle cells in the ventricular septum. Intramural coronary arteries are commonly narrowed due to the thickened arterial walls. It is commonly found in one or more closely related family members.

Idiopathic concentric left ventricular hypertrophy is probably related to H.C.M. in that unexplained hypertrophy is present. But unlike the former it does not exhibit the bizarre cellular disarray and the hypertrophy is concentric and not limited to the septum. Intramural coronary arteries are normal and it does *not* appear to be genetically linked.

Myocarditis is usually associated with systemic viral-like illness, it is acquired instead of congenital and is not infrequently identified as a cause of sudden death.

Sarcoidosis and right ventricular hypertrophy are rare but should be included for completeness.

PREDISPOSING FACTORS - VALVULAR

Valvular disease has been implicated in only about 3% of conditioned athletes who die suddenly during exercise (Waller)(10). This may reflect that patients with aortic valve stenosis have been identified prior to competition since this condition has such a widespread reputation for sudden death. Pulmonic stenosis theoretically also has a potential for sudden death.

Athletes with mitral valve prolapse *may* have a slightly increased risk of sudden death from arrhythmias. Most children and adolescents with this problem are asymptomatic. The precise role of M.V.P. in sudden death is unclear since data from the Framingham study suggests that the incidence of arrhythmias in patients with M.V.P. is no longer greater than that of the general population(13). It may be that symptomatic individuals with appreciable mitral regurgitation form a subset at higher risk and should be identified.

Ebstein's anomaly of the tricuspid valve consists of a distorted tricuspid valve. This often leads to variable degrees of right ventricular dysfunction. It is sometimes associated with conductive abnormalities such as Wolff-Parkinson-White syndrome.

PREDISPOSING FACTORS - AORTIC

Dilatation of the thoracic aorta due to cystic medial necrosis may develop in subjects with Marfan's Syndrome. This is an inherited disorder involving connective tissue. As the fewer medial elastic fibers in the aortic wall weaken, dissection, rupture and sudden death may occur.

PREDISPOSING FACTORS - CARDIOVASCULAR CONDUCTIVE

It is unusual for athletes to die suddenly without structural cardiovascular predisposition. Conductive variations have been described (James) (14) but usually are associated with some structural abnormality.

Electrocardiographic abnormalities such as bradycardia, second degree heart block and even AV dissociation have been reported in conditioned athletes but no data is available to link them to sudden death.

Idiopathic long QT syndrome is an uncommon hereditary condition where the QT interval is prolonged. It has been associated with tachy arrhythmias and ventricular fibrillation and with sudden death in adolescents and children. One variety of the syndrome is associated with deafness but there are no other physical findings specific for this hereditary syndrome.

DEATHS IN ALASKA - A SUMMARY

Eight cases of sudden unexpected deaths in Alaska age 6 to 30 years old which occurred between June, 1987 and July 1988, were reviewed and their causes of death summarized.

Three of the eight victims were found to have myocarditis, which proved to be the most common diagnosis.

Also represented were asthma (1), subarachnoid hemorrhage (1), aspiration of stomach contents (1), and ruptured aortic aneurysm (1). One case was diagnosed as having a spontaneous arrhythmia.

The one case that was exercise related was an 11 year old boy who died after collapsing in Karate class with a severe asthma attack.

The one spontaneous arrhythmia occurred in an individual with an enlarged heart but no known cardiac disease. There is no other information available about this case.

One can see from the types of these sudden deaths that the etiologies as defined in the larger studies are also represented here. Certainly, this small group does not reflect the same frequency as larger studies, but in general does reflect the types of sudden unexpected deaths.

POSSIBLE PREDICTORS

The basic challenge to physicians lies in the quest to enhance predictive probabilities of sudden death in our younger athletes. Understanding the medical conditions responsible for exercise-related sudden death is not enough. An increased awareness of potential sudden death is critical if those at higher risk are to be selected for more intensive medical study. Not only those doing pre-participation sports physicals, but also the coaches and trainers who are the direct observers of exercising athletes should be alert to these often subtle predictors.

Those found to be in high risk categories deserve a more complete and thorough cardio-pulmonary evaluation. Attempts should be made to delve more deeply into hereditary disease.

The following three areas will reveal the highest return when used with a careful physical evaluation.

(1.) Syncope:

In several studies, exercise associated syncope was relatively common (25%) for patients with sudden death especially in myocardial disease (16). Between 12 and 15% of normal adolescents experience such syncope. One must try to distinguish between the benign syncope and the more malignant exercise-associated type. Benign vasopressor syncope is usually not associated with exercise and occurs commonly in response to emotional stress or hot crowed rooms.

(2.) **Family History:**
A family history of sudden, unexpected nontraumatic death deserves a thorough evaluation. Most hereditary conditions including hypertrophic cardiomyopathy, Marfan's syndrome, and even prolonged QT interval may then be uncovered.

(3.) **Abnormal Cardiac Exam:**
Those individuals with abnormal cardiac evaluations on pre-competition sports physicals demand further evaluations. High risk findings would include at least arrhythmias, the murmurs of aortic, pulmonary stenosis, mitral or tricuspid regurgitation, and those that increase with Valsalva or exercise which may signal hypertropic cardiomyopathy.

ADDITIONAL TESTING

Echocardiography is an easily employed non-invasive testing method to help determine chamber size, septal size and valvular deformities.

Graded exercise stress testing can be used to evaluate arrhythmias and murmurs, as well as exercise tolerance per se. Twenty-four hour monitoring of the EKG is readily available in more communities. Other detailed studies such as coronary arteriography should be reserved for those at the greatest risk. Even this testing, however, is not completely without risk, nor does it always identify the problem lesions.

SUMMARY

In summary one can conclude that:

(1.) Those athletes with *structural* cardiovascular disease are at increased risk of sudden death during physical activity.

(2.) Risk of sudden death in all athletes may well be related to the type and intensity of *physical activity.*

(3.) Risk of sudden death is related to the *type* and *severity* of cardiovascular conductive disease.

Careful clinical assessment may help predict sudden death in some athletes, but not all of those at high risk. Observations by those on the field or court-side may be the key to recognizing those predisposing factors. It takes team work to sort out those few individuals at highest risk. Our vigilance coupled with an increasing awareness is our only hope of identifying those young athletes at high risk for sudden death.

ACKNOWLEDGEMENTS

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Results of an Indoor Air Pollution Investigation

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ABSTRACT

After the opening of a new elementary school in fall 1986, several members of the school's staff noticed symptoms they attributed to the workplace. An investigation by the Occupational Safety and Health Branch (OSHA) of the Alaska Department of Labor found no major health problems and concluded that fireproofing at the school may have caused a petroleum-like odor. In fall 1987, parents reported illness in their children that they attributed to school attendance. Subsequent epidemiologic investigation found that student and staff absentee rates were not increased and were similar to other schools in the district. Testing conducted by the National Institute for Occupational Safety and Health (NIOSH) confirmed that the petroleum-like odor was due to the fireproofing. Toxicologic information and the results of air sampling confirmed that no adverse health effects would be expected from the product. By working together with NIOSH, OSHA, the school district, and a consulting mechanical engineer, deficiencies in the school's ventilation system were discovered and immediately corrected with interim engineering changes. Only by bringing multiple agencies together and openly sharing findings with concerned parents and staff were the problems at the school resolved.

INTRODUCTION

During the past 30 years, considerable effort has been made and progress achieved toward reducing outdoor air pollution and chemical contaminants in the industrial environment. Although health hazards associated with at least one indoor air pollutant, carbon monoxide, have been known for thousands of years (1), indoor air pollution in general has been recognized as an important and complex public health concern for only the past several years (2). The construction of energy efficient buildings during the 1970s and 1980s led to concern that reduced ventilation might result in inadequate indoor air quality and stimulated research and investigation of building-related health complaints. For example, between 1971 and 1987, the National Institute for Occupational Safety and Health (NIOSH) responded to approximately 450 complaints of indoor air quality problems in office buildings (3).

In January 1988, the Section of Epidemiology of the Alaska Department of Health and Social Services began an investigation of illness thought to be caused by indoor air pollution at a recently opened elementary school in Wasilla, Alaska. For more than a year, the school district received complaints from parents and staff about poor air quality and possible building-related illnesses. Although the school district made extensive efforts to identify and correct potential problems, these efforts did not allay parental and staff perception that exposure in the building resulted in illness. This report describes the results of the investigation undertaken by the Section of Epidemiology of the Alaska Division of public Health.

BACKGROUND

In August 1986, a new elementary school opened in Wasilla, Alaska with approximately 400 students and 35 staff members. Beginning in January 1987, several teachers working on the first floor of the building began to experience a variety of symptoms which they felt were work-related. Symptoms included headache, abdominal pain, nausea, eye irritation, shortness of breath, dizziness, sore throat, and difficulty concentrating. The symptomatic teachers also noted a kerosene-like or petroleum-like odor in some of the classrooms and the first floor hallway. The school district requested the Labor Standards and Safety Division of the Occupational Safety and Health Branch (OSHA) of the Alaska Department of Labor to conduct a safety/health consultation. OSHA did not identify a specific causative agent for the odor, although they believed the source was fireproofing which was spayed above the first floor classrooms and hallway during construction.

The type of fireproofing used at the school has been widely used in the United States for at least 10 to 15 years, and is present in thousands of buildings (Jagnandan R. United States Mineral Products Company; Stanhope, NJ. Personal communication). Within Alaska, it was used in at least seven major projects, including office buildings, schools, a hotel, and a correctional facility (Englert J. Vertecs Corporation; Anchorage, AK. Personal communication). The product's composition is 97% silicates and calcium sulphate and 3% petroleum process oil. According to the manufacturer, the process oil is a chemically neutralized middle distillate petroleum which has been in use for more than 20 years. OSHA measured carbon dioxide (CO_2) in six first-floor classrooms and found most levels to be in the 500 to 600 parts per million (ppm) range. Two rooms had higher levels, 800+ ppm and 1,000 ppm. Since CO_2 levels >1,000 ppm are commonly accepted as an indication of an inadequate ventilation system, OSHA concluded that inadequate ventilation may have played a role in the reported symptoms.

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OSHA collected air samples for measurement of benzene, toluene, formaldehyde, short- and long-chain hydrocarbons, and other organics. All levels were substantially below the OSHA permissible exposure limits (PELs*) for industrial settings. For example, the concentration of formaldehyde (.01 ppm) was one one-hundredth of the permitted industrial exposure for this chemical (1.0 ppm).

In May and June 1987, the school district and the local Public Works Department investigated the ventilation system. After taking airflow measurements and resetting thermostats, they concluded that the system was operating as designed. Because OSHA felt the most likely source of the odor was the petroleum process oil contained in the fireproofing, during the summer of 1987 the school district raised the temperature and increased ventilation in the building in order to hasten the oil's evaporation ("off-gassing").

When school resumed in fall 1987, employees continued to complain of odor and building-related illness. In addition, several parents observed that their children had developed symptoms (including headache, dizziness, abdominal pain, cough, and runny and itchy eyes) which parents felt were school related. Complaints seemed to originate primarily from students in first-floor classrooms (kindergarten through third grade). In November 1987, OSHA completed an additional evaluation which documented abnormal air flow out of an exhaust (or return) air duct into a classroom. The odor in the return air was noted to be similar to the odor which was the source of the original complaints. OSHA recommended that the district apply a sealant over the fireproofing.

In December 1987, the fireproofing material was treated with a sealant. During January and February 1988, complaints of odor and illness continued. With increasing concern and anxiety, parents and staff questioned whether the school should remain open. At this point the Section of Epidemiology was invited to assist.

METHODS

We decided initially to investigate student and employee symptoms and absentee rates since this approach was the most efficient way to characterize illness and estimate the magnitude of the problem. We also investigated the toxicology of the fireproofing materials used at the school.

On-Site Interviews - To better characterize illness, all symptomatic employees were interviewed. In addition, mothers from the most concerned families were asked about their children's symptoms. We asked about symptoms which occurred during the period beginning when school opened in fall 1986 through February 1988.

Absenteeism - The school district provided information on student enrollment and absences at the study school and two comparable elementary schools in the district. The comparison schools were nearly the same size and were located in adjoining areas of the district. The building at one of the comparison schools was almost identical to the study school; the only significant differences being that different fireproofing and a somewhat different internal supporting structure were used at the comparison school.

Absentee rates were defined as the total number of absences in a specified interval divided by the number of student-days in the interval. Rates were calculated for school years 1986-87 and 1987 through the first week of February 1988. According to the district, absentee data during the first two weeks of each school year were unreliable. Accordingly, absence rates were calculated starting from the third week of the school year. Kindergarten absence rates were not studied since one of the comparison schools did not have kindergarten students. Absence rates by grade level and for each day of the week were calculated.

Employee sick-leave records were examined. For each employee at the study school and a comparison elementary school, the district provided a list of sick-leave used. Absences due to pregnancy, surgery, or chronic conditions (such as back injury) were excluded since these were considered unlikely consequences of possible ventilation problems or indoor air pollution at the worksite. Sick-leave use by first-floor and second-floor teachers at the study school were compared for three time periods (Fall 1986, Spring 1987, and Fall 1987) using analysis of variance (SAS Institute, Inc. Cary, NC).

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* No PELs for non-industrial settings exist. The use of industrial PELs was not meant to imply that industrial standards should be applied to school children and served only as a reference.

Toxicology - Since OSHA identified the fireproofing as the probable source of the odor, much attention at the school focused on this material. We requested toxicologic information on the fireproofing, the petroleum process oil contained in it, and the sealant from the manufacturers, the U.S. Food and Drug Administration (FDA), and NIOSH.

RESULTS

On-Site Interviews - Of the 35 school employees, 15 (and one parent volunteer) had symptoms which they felt were school-related. All 16 persons were interviewed, each reported noticing a petroleum-like odor at the school. The most frequent complaints were headache (13 persons), dizziness (11 persons), sneezing (9 persons), shortness of breath (10 persons), and sore throat (10 persons). Symptoms reported less often included itchy or runny eyes, cough, fever, impaired memory, disorientation, dry throat, and rash (Table 1). Of the 16 persons, 11 (69%) felt their symptoms were noticeably worse on school days as compared to weekends or other days off. Ten (63%) of the people interviewed regularly worked in the first-floor classrooms, two (12%) were assigned to the second-floor classrooms, and four (25%) did not work in classrooms on a regular basis. The greatest number of affected employees were on the first-floor (10/16), and most of the employees on the first-floor were symptomatic (10/11).

Seven mothers of eight pupils who expressed concern about possible school-related illness in their children were interviewed. According to the mothers, two children noticed an odor. Among the eight children, seven had either headache, cough, or sore throat, and six had abdominal pain. Less frequent symptoms were itchy or runny eyes, sneezing, fever, dizziness, diarrhea,

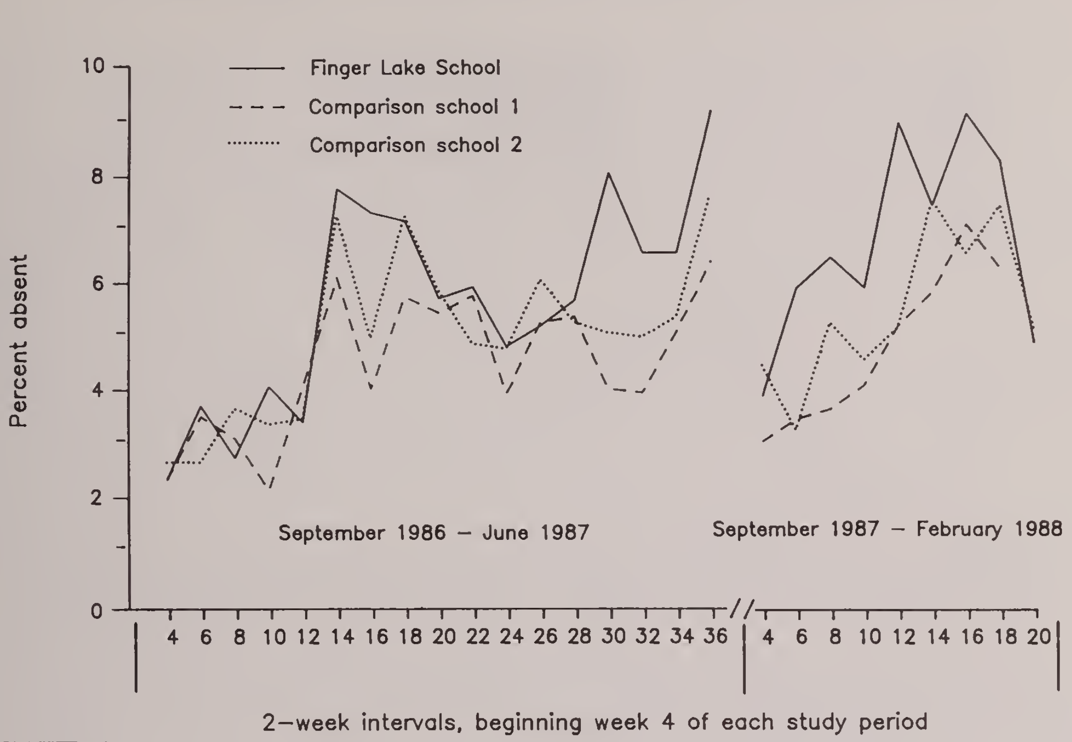
Table 1.
Symptoms Reported by Staff and Students
August 1986 - February 1988, Alaska

	Staff* (n=16)	Students* (n=8)	Total* (n=24)	Percent With Symptom*
Headache	13	7	20	83
Sore Throat	10	7	17	71
Cough	8	7	15	63
Dizziness	11	3	14	58
Sneezing	9	5	14	58
Abdominal Pain	6	6	12	50
Itchy Eyes	7	5	12	50
Shortness of Breath	10	1	11	46
Fever	6	3	9	38
Runny Eyes	3	5	8	33
Diarrhea	3	2	5	21
Burning Eyes	5	0	5	21
Rash	2	0	2	8
Sinus Problems	2	0	2	8
Weight Loss	0	1	1	4
Impaired Memory	1	0	1	4
Hoarseness	1	0	1	4
Queasiness	1	0	1	4
Stuffy Nose	1	0	1	4
Earache	0	1	1	4
Lethargy	0	1	1	4
Fatigue	1	0	1	4
Disorientation	1	0	1	4

*Total staff members = 35
Total students= 400
n = number of persons with symptoms attributed to the school.

Percent with symptom refers to the percent of persons with any school attributed symptom having a specific symptom.

Figure 1. Absenteeism at three Mat-Su Borough Schools, grades 1-6 combined, Alaska, September 1986 - June 1987 and September 1987 - February 1988



and lethargy (Table 1). With a single exception, all children had spent at least one school year in a first-floor classroom.

Absentee Rates and Sick-Leave Analysis - Absentee rates during school years 1986-87 and 1987-88 were low with only small differences between the study and comparison schools (Figure 1). For the 1987-88 school year, the student absentee rate at the study school was slightly higher than at the two comparison schools. The absentee rate was approximately the same for each of the first three grades at the study school (Figure 2) and rates for grades 4-6 were also similar (data not shown). The distribution of absences by day of the week at the study school was similar to the comparison schools with highest proportions of absences occurring on Mondays and Tuesdays and declining rates for the remainder of the week (Figure 3). For school year 1986-87, the mean number of employee absence days determined by sick-leave records at the study school (6.06 per employee) was similar to the comparison school (5.99 per employee). Likewise, for 1987-88, the mean number of absence days per employee at the study school (4.09) was similar to the comparison school (3.81 per employee). Sick-leave use for first-floor compared to second-floor teachers at the study school for 1986-87 and 1987-88 is shown in Table 2. The absentee rate for the 10 second-floor teachers (2.2 sick days per person) was the lowest of the six rates observed, however, a two-way analysis of variance of time period versus floor assignment showed no significant effect of either characteristic on absentee rates.

Toxicology - Requests to the product manufacturers for toxicologic information, illness reports, and technical assistance were generally referred to the legal rather than the medical or industrial hygiene section and responses sometimes took weeks to be received. Other than possible irritative effects to persons applying the fireproofing sealant, the manufacturers reported they were unaware of any human health problems related to the products involved. According to the manufacturer, the petroleum process oil qualifies as an "indirect food additive" by meeting the requirements of the FDA and may be used as a component in food packaging materials and lubricants contacting food (4). NIOSH research did not disclose any other human health threats from the products (Newman M., NIOSH; Cincinnati, OH. Personal communication).

INTERIM RECOMMENDATIONS AND ADDITIONAL FINDINGS

After completing the initial investigation, we prepared an interim report and made a presentation at a well-attended public meeting at the school. This gave parents and others a chance to learn about the various

Figure 2. Absenteeism at Finger Lake School, grades 1, 2, and 3, Alaska, September 1986 – June 1987 and September 1987 – February 1988

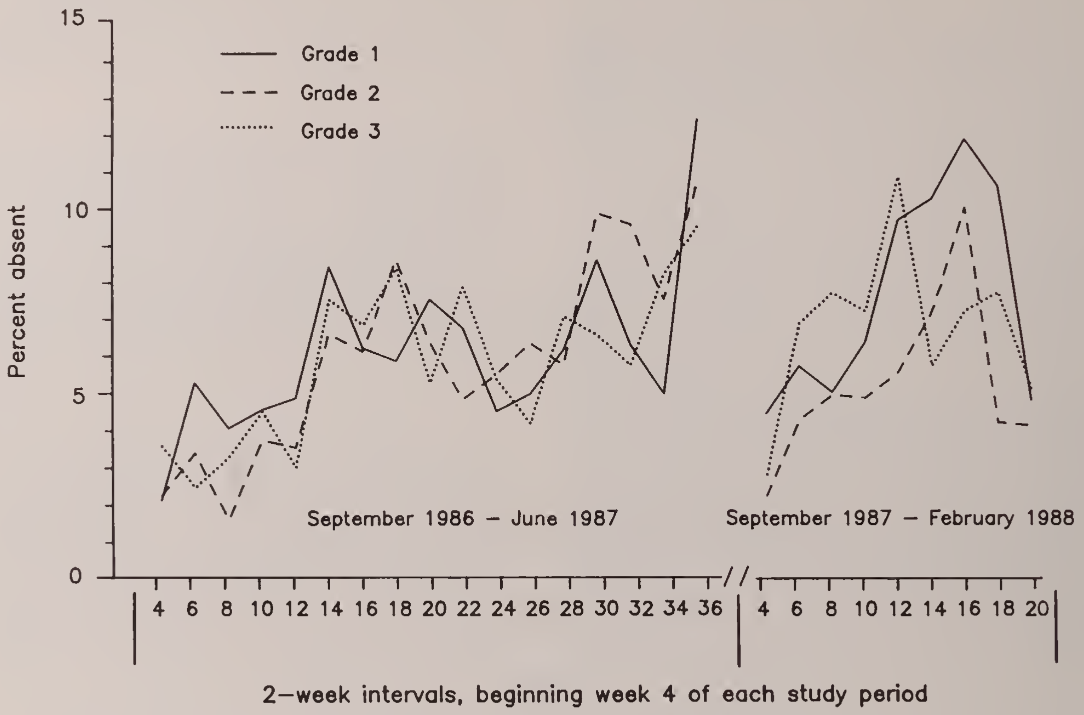
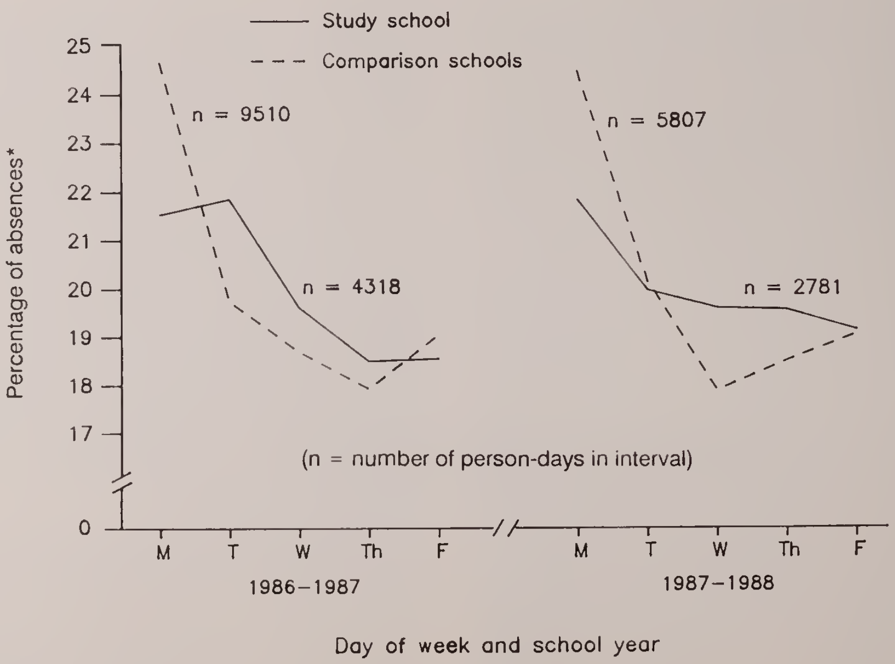


Figure 3. Percentage of absences by day of week for study school and two comparision schools, Alaska, 1986-1987



* The percentage of absences is corrected for the variable number of each day of the week in the two school years.

Table 2.
Absentee Rates for Teachers at Study School September 1986 to January 1988, Alaska

	Period 1 Sept. 86 - Dec. 86 (78 days)			Period 2 Jan. 87 - June 87 (99 days)			Period 3 Sept. 87 - Jan. 88 (104 days)		
	No. Sick Days	Teachers Assigned	Absentee Rate*	No. Sick Days	Teachers Assigned	Absentee Rate*	No. Sick Days	Teachers Assigned	Absentee Rate*
1st Floor	31.5	10	4.0	34.0	10	3.4	35.5	11	3.1
2nd Floor	17.0	10	2.2	34.0	10	3.4	36.5	12	2.9

$$\text{*Absentee Rate} = \frac{\text{No. Sick Days}}{\text{(No. of Teachers Assigned) x (No. of Days in Period)}} \times 100$$

activities that already had been completed, to voice their concerns, and to ask questions. We recommended that the school district obtain expert, independent consultation to evaluate the ventilation system and requested that OSHA collect additional samples to definitively identify the source of the petroleum-like odor at the school. In addition, we strongly recommended that a committee be formed to include concerned parents, the Section of Epidemiology, the local Public Works Department, the school district's medical advisor, OSHA, and school district officials in order to monitor progress of the investigation and effectiveness of control measures.

Subsequent evaluation of the ventilation system by a consulting mechanical engineer showed that airflow into classrooms was substantially below the standards recommended by the American Society of Heating Refrigerating, and Air-Conditioning Engineers (ASHRAE) (5). In addition, the engineer found, as OSHA had first documented, return air (which should go to an air-handling room) flowing back into some of the first-floor classrooms. The underlying causes for the inadequacies were described by the engineer and included design, construction, and operational deficiencies (Table 3).

Samples collected by OSHA and analyzed by NIOSH indicated that air sampled from areas noted to have the kerosene-like odor had the same series of organic chemicals (at very low concentrations) as both the petroleum process oil contained in the fireproofing and the gas evaporating from a sample of the fireproofing. Thus, we concluded that the odor was caused by the fireproofing. OSHA also collected multiple repeat air samples for quantification of a variety of organics and made numerous measurements of CO_2, humidity, temperature, and dust. Although temperature and humidity were both outside the normal comfort range, all potential contaminants (hydrocarbons, toluene, and formaldehyde) were substantially below the lowest standards (Table 4).

After these findings were discussed at meetings of the working committee which we recommended be established, the district implemented temporary changes to increase ventilation rates and stop backflow. In

Table 3.
Engineering, Mechanical and Construction Problems Identified at Study School, February 1988, Alaska

1. Return air ducts partially obstructed by constricted passages around doorways.
2. Supply fans unable to deliver recommended air flow.
3. Thin gauge material used in supply ducts subject to vibration and noise.
4. Fireproofing material sprayed inside return air ducts.
5. Ventilation system minimum airflows set below recommended standards for fresh air delivery.
6. Door of air-handling room frequently left open causing reduced flow through return air duct system.
7. Heating coils in air-handling unit unable to handle required volume of outside air.

Table 4
Results of OSHA* Sampling at Study School
February - March 1988, Alaska

Measurement	Result	Standard**	Comments
Long-chain hydrocarbons	0.047 mg/m^3	5.0 mg/m^3	mean of 6 samples from occupied rooms
N-Hexane	0.037 mg/m^3	180.0 mg/m^3	mean of 6 samples from occupied rooms
Other C6-C7 alkanes	0.092 mg/m^3	180.0 mg/m^3	mean of 6 samples from occupied rooms
Toluene	0.004 mg/m^3	375.0 mg/m^3	mean of 6 samples from occupied rooms
CO_2	578 ppm	1000 ppm	mean of 17 locations range: 430 - 710 ppm
Temperature	20.2°C	23-25°C (ref. 8)	mean of 72 measurements range: 17-22°C
Humidity	22.9% R.H.	40-60% (ref. 3)	mean of 72 measurements range: 11-29%
Formaldehyde	0.013 ppm	1.0 ppm	mean of 15 samples range : <.01 - .02 ppm
Dust	0.201 mg/m^3	5.0 mg/m^3+	mean of 13 samples range: <.07 - .969 mg/m^3 outside level: .102 mg/m^3

Notes:
* Collected by Alaska Department of Labor, OSHA. Measurements of hydrocarbons, n-hexane, and toluene done by NIOSH, Cincinnati, OH.
** Lowest published PEL or Threshold Limit Value.
+ Standard is PEL for respirable dust. OSHA PEL for total dust is 15 mg/m^3.

addition, the three most distressed teachers were transferred to other schools. Personnel at the school monitored the situation and as the district moved toward making permanent modifications to the ventilation system, complaints of odor and illness decreased.

DISCUSSION

Our investigation showed that student and employee absentee rates at the new elementary school were not substantially different than at two other schools in the district. This finding was extremely reassuring since some parents and teachers perceived that something at the school was making people seriously ill. There was no evidence in the interviews with teachers or mothers that effected persons had any specific illness or symptomatology. The range of symptoms was broad, nonspecific, and did not suggest any discrete toxic exposure. The observation that the absentee rate was higher earlier rather than later in each week indicated that illness was probably not caused by a sensitizing exposure at the school. Likewise, toxicologic information was reassuring that neither the fireproofing, petroleum process oil, nor sealant would cause any serious acute or chronic adverse health effects. Confirmation that the odor was due to the petroleum process oil removed all doubt about the source of the odor.

Ultimately, the specific cause or causes for the symptoms at the school could not be determined, however, there are two possibilities which might explain what happened: Symptoms were due to inadequate ventilation; i.e., "sick building syndrome." This syndrome was first described in the 1970s and is characterized by nonspecific complaints that cannot be attributed to any identifiable exposure. In tightly constructed buildings it is possible for poorly regulated humidity or temperature, inadequate fresh air supply, or accumulation of indoor air pollutants to lead to most of the symptoms reported at the school (2,6). Clearly, fresh air ventilation at the school was far below the minimum recommended by ASHRAE and this may have caused a sense of discomfort. Since temperature and humidity were less than optimal, this may also have contributed to the problem.

Secondly, the presence of an odor, and anxiety which might arise because the odor is thought to represent a potentially toxic exposure, could cause persons to attribute symptoms to an exposure at the school. Many of the reported symptoms might occur in the general population over any 12- to 18-month period even in the absence of a specific environmental exposure. Given the persistent, insidious odor, some staff members or parents may have concluded that something at the school caused the symptoms.

Probably, both of these factors played a role in the developments at the school. Because the odor was essentially eliminated by correcting the ventilation deficiencies, it was impossible to determine if the improved ventilation or the reduction of odor, or both, was responsible for the decrease in illness complaints.

This investigation required a multi-disciplinary, multi-agency effort which included representatives from the school district, OSHA, state health department, NIOSH, and a contracted mechanical engineer. Investigation of the problem and resolution of the complaints would have been difficult or impossible without this combining of expertise from wide-ranging, highly technical disciplines. Only by working cooperatively, and synergistically, were the problems overcome.

During this investigation, we requested all available toxicologic information and technical assistance from the manufacturers of the products involved. We also asked the manufacturers for reports of illness in workers or other exposed persons in other places in the United States. The responses we received were generally less than cooperative and our inquiries to health professionals were referred to the legal rather than the medical or industrial hygiene section. Requests for information that should have been responded to in hours or days were sometimes answered only after several weeks. The manufacturers were the only uncooperative participants in this multi-agency investigation.

Establishing open lines of communication between all persons involved in the investigation, and especially parents and staff at the school, was essential. Almost two complete school years elapsed between the time complaints first surfaced and this investigation identified serious ventilation deficiencies. During that period, rumor and concern at the school proliferated and communication from the school district was limited. By establishing a working committee which met on a regular basis as the investigation proceeded and recommendations were implemented, all parties were kept up-to-date on the progress of the investigation.

CONCLUSION

This investigation identified serious deficiencies in the ventilation system at an elementary school which first opened in fall 1986. Epidemiologic investigation could not confirm higher absentee rates for students or increased sick leave use among staff. An odor, which was determined to be due to a component in the fireproofing, was noticed at the school and the presence of this odor may have resulted in the perception that there was increased illness. Because energy efficient buildings are particularly common in locations where the climate is extreme, other buildings are likely to become the focus of health-related concerns in the future. Although NIOSH has published recommendations for evaluating indoor air quality problems (3), investigations may become quite complex and require assistance from local, state, or federal public health authorities. Engineering consultation, generally available only from the private sector, may also be needed. Building occupants will have considerable interest in the findings, recommendations, and control measures, if any, as an investigation proceeds and every attempt must be made to establish clear and open lines of communication. In many situations this may be effectively accomplished by forming a committee with representatives from all involved organizations as well as the building occupants.

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Laboratory Tests – False-positive tests for urobilinogen with Multistix® may occur during therapy with nizatidine.

Drug Interactions – No interactions have been observed between Axid and theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin. Axid does not inhibit the cytochrome P-450-linked drug-metabolizing enzyme system; therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur. In patients given very high doses (3,900 mg) of aspirin daily, increases in serum salicylate levels were seen when nizatidine, 150 mg b.i.d., was administered concurrently.

Carcinogenesis, Mutagenesis, Impairment of Fertility – A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice; although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups. The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a two-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy – Teratogenic Effects – Pregnancy Category C – Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in one fetus and at 50 mg/kg it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in one fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers – Studies conducted in lactating women have shown that <0.1% of the administered oral dose of nizatidine is secreted in human milk in proportion to plasma concentrations. Caution should be exercised when administering nizatidine to a nursing mother.

Pediatric Use – Safety and effectiveness in children have not been established.

Use in Elderly Patients – Ulcer healing rates in elderly patients are similar to those in younger age groups. The incidence rates of adverse events and laboratory test abnormalities are also similar to those seen in other age groups. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Clinical trials of nizatidine included almost 5,000 patients given nizatidine in studies of varying durations. Domestic placebo-controlled trials included over 1,900 patients given nizatidine and over 1,300 given placebo. Among reported adverse events in the domestic placebo-controlled trials, sweating (1% vs 0.2%), urticaria (0.5% vs < 0.01%), and somnolence (2.4% vs 1.3%) were significantly more common in the nizatidine group. A variety of less common events was also reported; it was not possible to determine whether these were caused by nizatidine.

Hepatic – Hepatocellular injury, evidenced by elevated liver enzyme tests (SGOT [AST], SGPT [ALT], or alkaline phosphatase), occurred in some patients and was possibly or probably related to nizatidine. In some cases, there was marked elevation of SGOT, SGPT enzymes (greater than 500 IU/L) and, in a single instance, SGPT was greater than 2,000 IU/L. The overall rate of occurrences of elevated liver enzymes and elevations to three times the upper limit of normal, however, did not significantly differ from the rate of liver enzyme abnormalities in placebo-treated patients. All abnormalities were reversible after discontinuation of Axid.

Cardiovascular – In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in two individuals administered Axid and in three untreated subjects.

CNS – Rare cases of reversible mental confusion have been reported.

Endocrine – Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to Axid. Impotence and decreased libido were reported with equal frequency by patients who received Axid and by those given placebo. Rare reports of gynecomastia occurred.

Hematologic – Fatal thrombocytopenia was reported in a patient who was treated with Axid and another H_2-receptor antagonist. On previous occasions, this patient had experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental – Sweating and urticaria were reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity – As with other H_2-receptor antagonists, rare cases of anaphylaxis following administration of nizatidine have been reported. Because cross-sensitivity in this class of compounds has been observed, H_2-receptor antagonists should not be administered to individuals with a history of previous hypersensitivity to these agents. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other – Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine administration have been reported.

Overdosage: Overdoses of Axid have been reported rarely. The following is provided to serve as a guide should such an overdose be encountered.

Signs and Symptoms – There is little clinical experience with overdosage of Axid in humans. Test animals that received large doses of nizatidine have exhibited cholinergic-type effects, including lacrimation, salivation, emesis, miosis, and diarrhea. Single oral doses of 800 mg/kg in dogs and of 1,200 mg/kg in monkeys were not lethal. Intravenous median lethal doses in the rat and mouse were 301 mg/kg and 232 mg/kg respectively.

Treatment – To obtain up-to-date information about the treatment of overdose, a good resource is your certified regional Poison Control Center. Telephone numbers of certified poison control centers are listed in the *Physicians' Desk Reference (PDR)*. In managing overdosage, consider the possibility of multiple drug overdoses, interaction among drugs, and unusual drug kinetics in your patient.

If overdosage occurs, use of activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. Renal dialysis for four to six hours increased plasma clearance.

PV 2096 AMP [013089]

Additional information available to the profession on request.

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[1] Data on file, Lilly Research Laboratories.

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Special Feature

HUMANA HOSPITAL AIR AMBULANCE PROGRAM IMPROVES ACUTE CARE ACCESS

The Air Ambulance program at Humana Hospital-Alaska is more than a medical transport service. Through its associations with an independent physician group, rural health care providers, commercial and charter aircraft operators, and the Municipality of Anchorage, the Air Ambulance program is helping to meet a variety of medical needs throughout rural Alaska.

Since its inception in 1986, the program has grown to include a dedicated twin-engine airplane, the medevac transport contract with Alaska Native Health Service, and direct airport access for airplanes to Humana Hospital.

The Air Ambulance dedicated aircraft, owned and operated by Alaska-based Peninsula Airways, is a Cessna Conquest capable of carrying two patients and two medical attendants. This twin-engine turboprop can fly above most adverse weather conditions and features a fully pressurized cabin for patient and crew comfort.

In May 1989, Humana Hospital signed a contract with Alaska Native Health Service to provide complete emergency medical escort services for critically ill or injured adult and pediatric patients from across the state. This agreement has given the Air Ambulance program extensive exposure to residents and employers located throughout rural Alaska and significantly increased the number of flights for the growing program.

An unique development for the Air Ambulance program is a taxiway agreement between the Municipality of Anchorage (Merrill Field) and Humana Hospital. This joint private/public venture creates a new level of access to one of Alaska's major medical centers and the emergency acute care settings.

The taxiway access eliminates the need for patient transfer to a ground ambulance, saving valuable time over the traditional use of Anchorage International Airport. This represents a tremendous joint health care venture between government and private industry.

Airplanes, as well as helicopters, now use this direct access from Merrill Field to Humana Hospital when time is a critical factor in delivery of acute hospital and trauma care.

Referring hospital and clinic-based physicians, as well as mid-level practitioners accompanying their patients to Anchorage, appreciate the continuity of care the independent physicians and staff at Humana facilitate when patients arrive at Humana Hospital via the Air Ambulance program.

In some cases patient referral or transfer does not require a dedicated medical aircraft dispatched from Anchorage. For instance, many patients transferred from the Kenai Peninsula arrive at Humana Hospital via the Alaska Flight Centre's Navajo Chieftain medevac plane, with Soldotna paramedics as the flight crew.

"Just recently I flew four very critical patients to Anchorage and time was of the essence," says Gordon Arteno, owner of the peninsula-based operation. "The combination of time saved by having the plane based here in Soldotna and the direct taxiway access to your hospital may very well have saved lives."

A significant percentage of Air Ambulance patients from Dutch Harbor arrive at Humana Hospital via scheduled Peninsula Airways flights. Dick Harding, director of operations for the commercial airline, says one reason is because weather conditions sometimes make landing at Dutch Harbor difficult.

Peninsula Airways has a 16-passenger Metroliner (twin-engine prop jet) in Dutch Harbor overnight. When weather prohibits an Anchorage-based medevac flight from landing in Dutch Harbor, the patient may be flown out on the scheduled Peninsula Airways flight departing Dutch Harbor in the morning.

Humana Hospital also calls on two other Alaskan firms, Security Aviation and Alaska Helicopters, for air transportation back-up. Security Aviation operates a Cessna Citation II jet, a twin-engine Conquest, and a smaller Navajo Chieftain. Alaska Helicopters operates several different types of rotor craft, with access to an AeroCommander.

Coordinating the various air transport alternatives to best meet each Air Ambulance patient's needs requires professional training and experience, as well as a familiarity with conditions in remote Alaskan clinics and villages. Working as a team to achieve this balance is the program's full-time coordinator, Becky Lundqvist, RN, and Medical Director Peter Hackett, M.D., an independent physician.

Through their supervision and coordination, the professional Air Ambulance staff has grown to 31 members, in addition to the independent physician support of Denali Emergency Medicine Associates. This staff of specially trained nurses and paramedics keeps a 24-hour on call crew on readiness status, giving them the capability of being airborne in a medically equipped aircraft within 60 minutes.

The medical flight crew for the Air Ambulance consists of 26 aero-medically trained critical care registered nurses and five flight paramedics. These Air Medical Crew Members (ACMs) receive on-going medical education by Drs. Peter Hackett and Don Hudson of Denali Emergency Medicine Associates, the independent physician group which provides 24-hour coverage in the Humana Hospital Emergency Room.

Dr. Hackett, who also serves as the Medical Director for the Air Ambulance program, is a certified specialist in emergency medicine. As a pulmonary physiologist, he has served as a NASA consultant and continues his high altitude research on Mt. McKinley. Dr. Hudson, an independent emergency room physician, is a medevac pioneer in Alaska and former Air Force flight surgeon.

Drs. Hackett and Hudson, along with their associates Drs. Mark Moeller (the first Air Ambulance Medical Director), Gil Dickie, Scott Sims, Brad Selden, and Valerie Rossetti, are independent physicians providing 24-hour, on-call coverage for the Air Ambulance program.

"We also try to be readily available to communities as a resource, giving the local health care providers access to on-going training, medical supplies and support that might be difficult for them to arrange locally," explains Dr. Hudson. "Occasionally the medical community can be difficult to access from the Bush so this accessibility is important."

Members of the independent physician group also rotate to several remote clinics as primary providers. "This gives us direct insight into community problems and allows us to learn who the leaders and problem-solvers are in that community so we can then help them to help their own community," Hudson says.

"Our intent is to develop good rapport with the local EMTs and other health care providers, fostering referral patterns and offering follow-up care and support to these people 24 hours a day, every day of the year," he says.

Humana Hospital-Alaska's Air Ambulance program represents another strong effort by Alaska's largest tax-paying acute care facility to provide quality medical service to Alaska. Affordable access to medevac service and acute hospital care has greatly improved since the Air Ambulance program started nearly three years ago. The benefactors are the people who live, work and play in Alaska.

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1. For the treatment of patients with hypokalemia with or without metabolic alkalosis, in digitalis intoxication and in patients with hypokalemic familial periodic paralysis. If hypokalemia is the result of diuretic therapy, consideration should be given to the use of a lower dose of diuretic, which may be sufficient without leading to hypokalemia.

2. For the prevention of hypokalemia in patients who would be at particular risk if hypokalemia were to develop, e.g., digitalized patients or patients with significant cardiac arrhythmias.

The use of potassium salts in patients receiving diuretics for uncomplicated essential hypertension is often unnecessary when such patients have a normal dietary pattern and when low doses of the diuretic are used. Serum potassium should be checked periodically, however, and if hypokalemia occurs, dietary supplementation with potassium-containing foods may be adequate to control milder cases. In more severe cases, and if dose adjustment of the diuretic is ineffective or unwarranted, supplementation with potassium salts may be indicated.

CONTRAINDICATIONS Potassium supplements are contraindicated in patients with hyperkalemia since a further increase in serum potassium concentration in such patients can produce cardiac arrest. Hyperkalemia may complicate any of the following conditions: Chronic renal failure, systemic acidosis such as diabetic acidosis, acute dehydration, extensive tissue breakdown as in severe burns, adrenal insufficiency, or the administration of a potassium-sparing diuretic (e.g., spironolactone, triamterene, amiloride) (see **OVERDOSAGE**).

Controlled release formulations of potassium chloride have produced esophageal ulceration in certain cardiac patients with esophageal compression due to enlarged left atrium. Potassium supplementation, when indicated in such patients, should be given as a liquid preparation or as an aqueous (water) suspension of K-DUR (see **PRECAUTIONS**; **Information for Patients**, and **DOSAGE AND ADMINISTRATION** sections).

All solid dosage forms of potassium chloride are contraindicated in any patient in whom there is structural, pathological (e.g., diabetic gastroparesis) or pharmacologic (use of anticholinergic agents or other agents with anticholinergic properties at sufficient doses to exert anticholinergic effects) cause for arrest or delay in tablet passage through the gastrointestinal tract.

WARNINGS Hyperkalemia (see **OVERDOSAGE**)—In patients with impaired mechanisms for excreting potassium, the administration of potassium salts can produce hyperkalemia and cardiac arrest. This occurs most commonly in patients given potassium by the intravenous route but may also occur in patients given potassium orally. Potentially fatal hyperkalemia can develop rapidly and be asymptomatic. The use of potassium salts in patients with chronic renal disease, or any other condition which impairs potassium excretion, requires particularly careful monitoring of the serum potassium concentration and appropriate dosage adjustment.

Interaction with Potassium Sparing Diuretics—Hypokalemia should not be treated by the concomitant administration of potassium salts and a potassium-sparing diuretic (e.g., spironolactone, triamterene or amiloride) since the simultaneous administration of these agents can produce severe hyperkalemia.

Interaction with Angiotensin Converting Enzyme Inhibitors—Angiotensin converting enzyme (ACE) inhibitors (e.g., captopril, enalapril) will produce some potassium retention by inhibiting aldosterone production. Potassium supplements should be given to patients receiving ACE inhibitors only with close monitoring.

Gastrointestinal Lesions—Solid oral dosage forms of potassium chloride can produce ulcerative and/or stenotic lesions of the gastrointestinal tract. Based on spontaneous adverse reaction reports, enteric coated preparations of potassium chloride are associated with an increased frequency of small bowel lesions (40-50 per 100,000 patient years) compared to sustained release wax matrix formulations (less than one per 100,000 patient years). Because of the lack of extensive marketing experience with microencapsulated products, a comparison between such products and wax matrix or enteric coated products is not available. K-DUR is a tablet formulated to provide a controlled rate of release of microencapsulated potassium chloride and thus to minimize the possibility of a high local concentration of potassium near the gastrointestinal wall.

Prospective trials have been conducted in normal human volunteers in which the upper gastrointestinal tract was evaluated by endoscopic inspection before and after one week of solid oral potassium chloride therapy. The ability of this model to predict events occurring in usual clinical practice is unknown. Trials which approximated usual clinical practice did not reveal any clear differences between the wax matrix and microencapsulated dosage forms. In contrast, there was a higher incidence of gastric and duodenal lesions in subjects receiving a high dose of a wax matrix controlled release formulation under conditions which did not resemble usual or recommended clinical practice (i.e., 96 mEq per day in divided doses of potassium chloride administered to fasted patients, in the presence of an anticholinergic drug to delay gastric emptying). The upper gastrointestinal lesions observed by endoscopy were asymptomatic and were not accompanied by evidence of bleeding (Hemoccult testing). The relevance of these findings to the usual conditions (i.e., non-fasting, no anticholinergic agent, smaller doses) under which controlled release potassium chloride products are used is uncertain; epidemiologic studies have not identified an elevated risk, compared to microencapsulated products, for upper gastrointestinal lesions in patients receiving wax matrix formulations. K-DUR should be discontinued immediately and the possibility of ulceration, obstruction or perforation considered if severe vomiting, abdominal pain, distention, or gastrointestinal bleeding occurs.

Metabolic Acidosis—Hypokalemia in patients with metabolic acidosis should be treated with an alkalinizing potassium salt such as potassium bicarbonate, potassium citrate, potassium acetate, or potassium gluconate.

PRECAUTIONS General: The diagnosis of potassium depletion is ordinarily made by demonstrating hypokalemia in a patient with a clinical history suggesting some cause for potassium depletion. In interpreting the serum potassium level, the physician should bear in mind that acute alkalosis per se can produce hypokalemia in the absence of a deficit in total body potassium while acute acidosis per se can increase the serum potassium concentration into the normal range even in the presence of a reduced total body potassium. The treatment of potassium depletion, particularly in the presence of cardiac disease, renal disease, or acidosis requires careful attention to acid-base balance and appropriate monitoring of serum electrolytes, the electrocardiogram, and the clinical status of the patient.

Laboratory Tests: When blood is drawn for analysis of plasma potassium it is important to recognize that artifactual elevations can occur after improper venipuncture technique or as a result of in-vitro hemolysis of the sample.

Drug Interactions: Potassium-sparing diuretics, angiotensin converting enzyme inhibitors (see **WARNINGS**).

Carcinogenesis, Mutagenesis, Impairment of Fertility: Carcinogenicity, mutagenicity and fertility studies in animals have not been performed. Potassium is a normal dietary constituent.

Pregnancy Category C: Animal reproduction studies have not been conducted with K-DUR. It is unlikely that potassium supplementation that does not lead to hyperkalemia would have an adverse effect on the fetus or would affect reproductive capacity.

Nursing Mothers: The normal potassium ion content of human milk is about 13 mEq per liter. Since oral potassium becomes part of the body potassium pool, so long as body potassium is not excessive, the contribution of potassium chloride supplementation should have little or no effect on the level in human milk.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS One of the most severe adverse effects is hyperkalemia (see **CONTRAINDICATIONS, WARNINGS,** and **OVERDOSAGE**). There have also been reports of upper and lower gastrointestinal conditions including obstruction, bleeding, ulceration, and perforation (see **CONTRAINDICATIONS** and **WARNINGS**).

The most common adverse reactions to oral potassium salts are nausea, vomiting, flatulence, abdominal pain/discomfort, and diarrhea. These symptoms are due to irritation of the gastrointestinal tract and are best managed by diluting the preparation further, taking the dose with meals or reducing the amount taken at one time.

OVERDOSAGE The administration of oral potassium salts to persons with normal excretory mechanisms for potassium rarely causes serious hyperkalemia. However, if excretory mechanisms are impaired or if potassium is administered too rapidly intravenously, potentially fatal hyperkalemia can result (see **CONTRAINDICATIONS** and **WARNINGS**). It is important to recognize that hyperkalemia is usually asymptomatic and may be manifested only by an increased serum potassium concentration (6.5-8.0 mEq/L) and characteristic electrocardiographic changes (peaking of T-waves, loss of P-waves, depression of S-T segment, and prolongation of the QT-interval). Late manifestations include muscle-paralysis and cardiovascular collapse from cardiac arrest (9-12 mEq/L).

Treatment measures for hyperkalemia include the following:

1. Elimination of foods and medications containing potassium and of any agents with potassium-sparing properties.
2. Intravenous administration of 300 to 500 mL/hr of 10% dextrose solution containing 10–20 units of crystalline insulin per 1,000 mL.
3. Correction of acidosis, if present, with intravenous sodium bicarbonate.
4. Use of exchange resins, hemodialysis, or peritoneal dialysis.

In treating hyperkalemia, it should be recalled that in patients who have been stabilized on digitalis, too rapid a lowering of the serum potassium concentration can produce digitalis toxicity.

Caution Federal law prohibits dispensing without prescription.

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ALASKA
PUBLIC HEALTH ASSOCIATION

News from the Alaska Public Health Association

President's Message -- Susan Jones, R.N.,M.N.

This summer has been exciting and important for ALPHA. Representatives traveled to Alyeska and Washington, D.C. to talk with legislators and APHA affiliates about views on health issues, activities throughout the nation by APHA affiliates on legislative programs, membership development, revenue development, Year 2000 Objectives, and activities resulting from the Institute of Medicine report, *"The Future of Public Health."* Your president and President-elect, Brian Saylor, found that one of the more interesting results of attending the 1988 Affiliate President-elect Meeting in Washington, D.C. in July was the opportunity to compare our organization's activities with those from other states. Many of the states are focusing their activities on the Institute of Medicine report. Some states have established Blue Ribbon Panels to review the report and prepare a response. Papers responding to the report will be presented at the Annual APHA October meeting in Chicago. The APHA met in early May and will meet again in September to review the report and plan for effective action. A summary of their activities can be found in the Summer 1989 PROFESSIONAL AFFAIRS APHA newsletter.

"The Year 2000 Health Objectives" are a set of measurable, national health targets that have been sent to ALPHA for review and comment. APHA notes the importance of affiliate comments to help develop a single position on the "Objectives." This will be submitted to the Office of Disease Prevention and Health Promotion of the Public Health Service. At the July affiliate many states reported incomplete work on the Objectives for 1990, but dedicated their organization to help write clear and concise objectives for "Year 2000." If you are interested in reviewing the draft, please contact either myself or Brian Saylor.

Health issues that were discussed with Senator Ted Stevens and his aide, Senator Murkowski's aide and Congressman Don Young over the past two months were:

The APHA request for $172 million for the sexually transmitted disease program to control and prevent STDs as an important component in preventing transmission of AIDS.

Support for APHA request for the CDC Injury Prevention and Control Program to allow CDC to strengthen its integrated, multi-disciplinary approach to injury prevention with the goal of reducing mortality, morbidity, disability and cost associated with intentional and unintentional injuries.

Support for ALPHA's request for $139,000 to develop an Injury Prevention Resource Center to serve as a clearing house and be a central point for the collection of information on current injury prevention activities and available grants, and to provide training.

Support for the APHA request for the CDC immunization program ($218.5 million) which would support: continued immunizations at current levels; vaccine stockpile; activities associated with measles outbreaks; and the hepatitis B virus screening program.

Support for continued funding to those families that are marginally eligible for financial assistance for health services. In planning for the unemployed oil spill workers, Alaska may see an increase in the number of families who need the services but are temporarily not eligible. There may also be an increase in the number of homeless especially in the Anchorage area.

"Getting ALPHA's Future in Focus" is the theme of this fall's executive board retreat. Last year under Betsy Nobmann's able direction we set the path for ALPHA to follow. We changed the constitution and bylaws to support the purpose of ALPHA to "advocate for the right to health for all Alaskans through leadership in effective health policy, education and recognition of accomplishments." This year we want to have our yearly goals and activities be directly reflective of this purpose. We also would like your input and ideas on past and future activities. Please contact any board member and voice your opinion.

The establishment of the ALPHA Alaska Health Fund will be discussed during the fall retreat. The purpose of this fund will be to promote and support educational and professional growth activities. Criteria on who and how the fund will be used is in draft form and ready for board discussion. If you are interested in the discussion or have ideas, contact a board member.

A reminder to all: The BROWN BAG lunches are starting again. Use these to focus local health issues and develop resolutions for the annual meeting.The annual meeting will be held in Whitehorse, YT during the Symposium on Circumpolar Health in May 1990.Our membership is up to 161. Make December your month to recruit a new member.

The Future of Public Health in Alaska

Brian Saylor, PhD, MPH
Jennifer Christian, MD, MPH

Health care professionals in Alaska should take careful note of the warnings contained in *The Future of Public Health* (1). The report, published by a prestigious committee of the Institute of Medicine, paints a bleak picture of the state of public health in the United States. Based on a survey of state and local health departments, *The Future of Public Health* is a call for reevaluation of the role of public health in modern times and conditions. This article examines a couple of recommendations contained in the report and suggests how they may apply to the State of Alaska.

The overall findings of the report are clear: public health professionals in the United States have lost sight of their goals and allowed the public health system to fall into disarray. They have lost the zeal which produced the dramatic gains of the 19th and early 20th centuries. Satisfied with maintaining classic public health functions such as maternal and child health and basic sanitation, public health professionals have not claimed leadership of programs which combat the new "diseases of civilization" such as AIDS and toxic substances in air, water, and food.

The report notes two possible reasons for the abdication of leadership by the public health system. First, the public health community's mission has not had a dynamic evolution, but rather has been frozen in time. This problem is reflected in the disjointed and antiquated patchwork of national, state and local public health laws, regulations and policies. Second, the educational system has continued to train technicians, instead of developing a corps of skilled professional leaders to provide, manage, evaluate and develop new public health services. Both these reasons for the default of public health at a national level are especially relevant to Alaska.

The team which prepared *The Future of Public Health* did not visit Alaska. If it had, the overall description of the disarray of public health in our country may have been far more emphatic. Alaska's public health service delivery system is beset by baffling jurisdictional, organizational and financial obstacles to the provision of needed public health services to Alaskans. A policy vacuum and divided leadership has led to inconsistent allocation of resources, and blunted effectiveness.

The Future of Public Health recommends that existing state laws and policies be updated to broaden the mandate of public health agencies to include emerging threats to the public health. Rewriting laws and policies is also intended to accomplish other goals which are particularly important in Alaska.

First is the definition of those public services which are essential to promoting the public health and welfare of Alaskans, the funding of which is rightfully the responsibility of government. The task of defining these essential or "core" services was assigned to states and communities by *The Future of Public Health*. Currently, the Municipality of Anchorage Health and Human Services Commission is actively wrestling with this project.

A second major goal is the revision of the process by which public health powers are delegated from the state to localities. In most areas of the U.S., states delegate powers to county or local health departments. In Alaska, the law provides that public health services be directly delivered by state agencies, or units of local government which vote to assume health powers and are called Health Districts. There are only two such districts in Alaska (The Municipality of Anchorage and the North Slope Borough).

However, other agencies are now providing basic public health services through contracts with the state government, an arrangement inconsistent with state public health law. Most of these contracts are being awarded to Native Regional Health Corporations which are serving as quasi-governmental agencies. Questions have been raised about accountability for public funds, enforcement of contract provisions, and appropriate local fiscal support.

Another major recommendation in *The Future of Public Health* emphasized the need to strengthen the education of public health workers. In Alaska, this has a unique focus. Our University system needs to expand its ability to provide training in health and human services for personnel who will serve in rural Alaska. Historically, turnover in rural public health service has been high. Native regional corporations continue to assume responsibility for an ever-increasing proportion of health care services delivery. With reduced staff supplied by federal agencies, the Native regional corporations must attract and retain skilled nurses, sanitarians, counselors, administrators, and researchers.

The best candidates may come from local service areas. Alaskan students would be best served if the university system developed additional educational tracks in health sciences aimed at the needs of rural Alaskan communities. Educational subsidies from the State or Regional Corporations or expanded student loan forgiveness programs for health sciences students would probably be required to increase the pool of qualified professionals, especially in rural areas.

The Future of Public Health described a public health system in disarray. The failings outlined in the report are especially visible in Alaska. Many other issues and recommendations contained in the report make it "must reading" for Alaska health care professionals. For the future of public health in our state, we must develop a thoughtful response.

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[1] *The Future of Public Health*, Division of Health Care Services, Institute of Medicine, National Academy Press, Washington, D.C., 1988.

Drs. Christian and Saylor are with the Municipality of Anchorage Department of Health and Human Services. Dr. Saylor is President-elect of the Alaska Public Health Association. Dr. Christian is Secretary-Treasurer of the Alaska State Medical Association.

Editor's Note: Copies of *The Future of Public Health* are available from the Alaska Public Health Association.

HEALTH ISSUES COMMITTEE REPORT

This summer, the ALPHA Health Issues Committee met with legislators to discuss specific pieces of legislation of importance to the Association and began a review of *The Best of Care*, a report produced by the Governor's Interim Commission on Health Care.

Senator Sturgeluski discussed Senate Bill 3, which promotes boating safety. While marine accidents are one of the largest single causes of injury and death in Alaska, there does not appear to be much support for the passage of this bill. Rural legislators remain unconvinced that the bill will befit their constituencies. The Senator suggested that ALPHA members, particularly those who live in rural areas, contact their legislators to help promote the bill's passage.

Senator Sturgeluski is also supporting an Arctic Research Commission which will conduct research in health issues as they relate to the development of a comprehensive arctic health policy.

During a meeting with Representative Johnny Ellis, we learned that the Senate version of the seat belt legislation (SB 98) is still alive. He urged the Association to support the bill. Representative Ellis is supporting House Bill 10 which amends the student loan program to forgive the loans of students who pursue health degrees. The objective of the legislation to encourage health professionals to practice in Alaska, and thus help relieve some of the health manpower shortage in our state.

A subcommittee of the Health Issues Committee has begun a review of *The Best of Care* report. A resolution which referred to the report was not approved during the ALPHA Health Congress primarily because the membership was not familiar with its contents. In the coming months, the subcommittee will ascertain the status of the recommendations, and decide which of the recommendations still requiring action should be supported by ALPHA.

This fall, the Health Issues Committee expects to sponsor a series of public forums with legislators who are interested and influential in priority health care legislation.

"THE CIRCLE OF HOPE"

STATEWIDE HEALTH CONFERENCE

Ending an 8-year drought since the last statewide conference, the Alaska Native Health Board announced its plans for a winter conference February 6-8, 1990 at the Captain Cook Hotel in Anchorage.

Discussions and workshops will address the long expressed needs of rural and urban Alaska Native health care providers. Topics will cover problems associated with village providers, (community health aides, village public safety officers and community health representatives), such as training, pay and work condition. Local management of health care programs under the self determination (P.L. 93-638) will be explored. Issues of child abuse and neglect will be discussed as well as causes of high morbidity/mortality from injuries, suicide and homicide.

The conference will provide an opportunity to identify positive community programs and special projects that are attempting to reduce substance abuse and associated family problems experienced by Alaska Natives and to share their successes, thus expanding the "Circle of Hope," this year's convention theme. For further information, please contact June Degnan, Conference Coordinator, at (907) 337-0028.

Upcoming Events

- ALPHA Board Retreat, Wasilla, September 23-24, 1989
- 117th Annual American Public Health Association meeting, Chicago, October 22-26, 1989
- APHA Alaska Breakfast, Chicago, October 25
- ANHB "The Circle of Hope" Anchorage, February 6-8, 1990
- 8th International Congress on Circumpolar Health Conference, Whitehorse, YT, May 20-25, 1990

MEETING BETWEEN ALPHA AND SENATOR STEVENS

Tom Nighswander, MD, MPH

This year has provided ALPHA with the most extensive contact that we have had with any congressional office in our history. Senator Stevens and portions of his Washington staff were in Alaska the week of August 21. A private meeting was held with his health affairs staffer, Jane Rosenquist, Betsy Nobmann, Carl Hild and myself. I represented the Board at the general meeting which Senator Stevens had with representatives from the health community at Alyeska. Susan Jones also had an opportunity to meet with the Senator's staff when she was in Washington, D.C. in June. Carl Hild, Betsy Nobmann and myself met the Senator last December.

Highlights of our discussions include the Senator's support for an ALPHA sponsored injury control resource center and clearing house. The funding mechanisms are not clear, but both the Senator and Jane Rosenquist, Senator Steven's staff person in charge of health affairs, were very encouraging that this could happen. He pledged to increase financial support for the community health aide program which he considers partly his own program. He has sponsored additional funding for the injury control program for the Centers for Disease Control. He has been particularly concerned about the tightening of the eligibility for VA benefits. He mentioned his concern for issues of long range care of the elderly and was going to introduce the issue of the problem of caring for young children with chronic disease requiring institutional management.

However, he is very pessimistic about great amounts of additional monies coming from the Federal Government for Health and Human Services nationally and to the local level. He reiterated the problems with trying to control the national debt and pointed to the second largest budget item on the federal budget debt servicing, second only to defense.

My impressions were that his office is always willing to try to make the federal bureaucracy work and does not at all mind responding to those kinds of requests.

He is cost oriented and in response to a number of the proposals would always talk about not only the price tag but where savings might occur.

As I listened to the problems that people mentioned: survival of rural hospitals, the uninsured, escalating costs of care, reimbursement rates, etc., the system clearly is in trouble. However, his response, which probably is representative of Congress, is to try to fix each part and not deal with the system as a whole. He did mention that he and some of his colleagues were briefed by a group of Canadian health administrators and professionals who talked about the advantages of the national health program in Canada. He said it generated some interest and enthusiasm among his colleagues but he thought there were hidden real dollar costs in the system that were not identified.

Our meetings have been very instructive and helpful. I was impressed with Jane Rosenquist as being bright, sensitive to the issues, and also someone who had done her homework. She does have Alaskan roots. For any of your concerns about health affairs, I would send them directly to her. The Senator likes to hear from his constituents and he likes to be able to respond.

Brown Bag Lunches

The Brown Bag Lunch program offers a monthly forum for discussion of pertinent public health issues. Thus far, Anchorage and Fairbanks have established agendas for the year and both Bethel and Juneau are in the planning stage.

The Fairbanks Brown Bag Lunch is being chaired by Cheryl Kilgore and will be held on the second Tuesday of each month at the Fairbanks Health Center from 12:15-1:00 p.m.

The Anchorage Brown Bag Lunch is being chaired by Bert Hall and will be held the third Thursday of each month in the 4th floor conference room at the Anchorage Municipal Health Department. The luncheon begins at 11:30 a.m. with an informal get together and an opportunity to network with colleagues. The speaker will begin at 12:10, allowing for everyone to get back to work by 1:00. A catered meal (i.e. sandwich, etc.) will be available for those who wish to purchase it. If any other brown bag lunch sites have been established, please let the newsletter editor know so we can announce it in the next publication.

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History of Medicine in Alaska

WILLIAM MASSIE WHITEHEAD - 1905 - 1966

Virginia Whitehead Breeze wrote the following article for the Quarterly, a newsletter for the University of Virginia Medical School Class of 1930. It appeared in April 1987.

There is no way to capture the style and loving perceptions of Virginia Breeze. So with her permission, it is reprinted here. The article is followed by additional material about Dr. William M. Whitehead gathered from an interview with her.

William Massie Whitehead was born in Lovingston, Virginia, October 21, 1905, the fifth of seven children whom Susan Massie Whitehead and Stuart Baldwin Whitehead reared to adulthood at the family home, "Edge Hill," a large white-frame house that grew appropriately to accommodate five sons and two daughters. He was graduated from E.C. Glass High School in Lynchburg in 1923 and noted at the time that his greatest ambition was to manage the Salvation Army. His high school classmates voted him the biggest bonehead and the most athletic boy during the second semester of his senior year.

From E.C. Glass, Bill Whitehead went on to the University of Virginia to study medicine. My family has no record of his scholastic accomplishment from those years but we do have photographs that show him enjoying the good campus life. "Your mother belonged to Phi Beta Kappa and I belonged to Kappa Beta Phi," he told us in later years. "What was Kappa Beta Phi?" we would ask, knowing and anticipating his reply. "Both are scholastic honorary societies, only one is at the top and the other, at the bottom," he would say without a trace of regret.

It was after graduating from medical school in 1931 that Bill began the greatest adventure of his life: he moved west to Seattle to intern under the tutelage of Dr. James Tate Mason, University of Virginia Medical School Class of 1905, at the Virginia Mason Hospital, a private facility named for Dr. Mason's daughter. When his training was complete in 1932, Dr. Whitehead joined the American Mail Line as a ship's physician, traveling to China, Japan, Hong Kong and the Philippines. He then established a private practice in Seattle.

In 1934 he decided to move to Wrangell, Alaska, a small town, located on an island in Alaska's southeast "Panhandle." Why Alaska? It was, as it turned out, a decision of lasting importance but like many such decisions it was made for a simple, practical reason. "I had to eat," he would tell us later. "This was during the depression and I wasn't eating in Seattle." A small medical practice, already established, awaited him.

Young Dr. Whitehead became an immediate celebrity in Wrangell. A sociable bachelor with southern charm, he caught the eye of the town's comely young ladies but one in particular he liked in return: Dorothy Helene Johnson, half Norwegian, half Swedish, a statuesque beauty who looked a bit like Ingrid Bergman. She was also bright, an honors graduate from Whitman College in Washington State with a degree in economics and a teacher's certificate. "She chased me up a totem pole and wouldn't let me down until I said yes" he like to tell us. They were married at St. Phillip's Episcopal Church in Wrangell on September 23, the year he arrived.

Through the years the practice of medicine in Alaska has challenged the minds and resources of its physicians. Only very recently has modern medicine arrived in the vast north territory that historians once called "Seward's Folly." Alaska's physicians did the best they could with what they had to work with. In my father's case, in 1934, that meant, for instance, trying to save a premature baby by putting him in a shoe box heated by a light bulb because there was no incubator in Wrangell. "Hell, that kid grew up to be the best basketball player the town ever had," he liked to tell us. Fortunately Alaska seemed to attract outstanding members of the profession who apparently felt a sense of adventure about their decision to come or were determined to make a difference professionally in a frontier environment. There was, also, the opportunity to hunt, fish and "get away from it all," and finally, there was the lure of financial success though pioneer Alaska doctors were not reimbursed with pokes of gold.

On July 4, 1935, Dr. W.J.B. McAuliffe died in his office in Juneau, Alaska. The following month, Bill Whitehead moved 150 miles north from Wrangell to the territory's capital, which had a population of 7,500, to take over the practice. He located upstairs in the Shattuck Building on Seward Street and kept evening office hours and made himself available on weekends to build up his practice. On August 26, 1936, he formed the Juneau Clinic with three other Juneau general practitioners: Dr. W.W. Council, C.C. Carter, and W. P. Blanton. Their offices in the Goldstein Building burned in a fire that destroyed that building in February, 1939 and were then relocated above the Alaska Laundry on Juneau's South Franklin Street. The clinic stayed in that location, though in remodeled quarters, until after Dr. Whitehead's death. In 1965, however, he established a clinic on Willoughby Avenue under his own name and was in full-time practice there when he died, November 12, 1966.

Over the years, Bill and Dorothy Whitehead became active, respected members of the Juneau community. They reared a large family: four daughters and a son. They devoted hours and hours of their time to the activities of the Holy Trinity Episcopal Church, serving on the vestry and the altar guild and entertaining members of the far-flung statewide clergy in our family home. They raised flowers and vegetables at our summer cabin on Lena Beach, 15 miles north of Juneau. "Your father should have been a farmer," our mother would say as she watched him tenderly bunch up vegetables for neighbors and friends. Remembering his Virginia childhood he planted corn every spring in his greenhouse. It grew only a few inches high but he kept on planting it. He wore homegrown pansies as a boutonniere and nursed rose bushes through the long Alaskan winters by storing them in the concrete air-raid shelter he built in the backyard in 1941. ("Dr. Whitehead is ready for war," heralded *Life Magazine* in a photo spread on Alaska's defense at the time.)

About his medical practice I would say that he chose to make it a giving profession, which I believe, is the professional goal of most doctors. He located his family across the street from Juneau's Catholic hospital so he could get there quickly if called from home at odd hours. From our open bedroom windows we could hear him yell, "Push, push," to a women in labor in St. Ann's delivery room at night. He kept office hours, always, on Saturdays. "These young doctors think people get sick only on Mondays through Fridays,", he would grumble. As the town's obstetrics expert he willingly assisted other doctors with their own deliveries and he enjoyed assisting the chief surgeon, Dr. Carter, in the operating room. He also had a yearning to be an inventor and came up with a quick, almost painless way to pierce ears and a chest rub he called "Whitehead's Ointment #2."

He was never an intellectual and he had little use for doctors who talked in philosophical or scientific abstracts. He liked to visit with people, discuss their ailments, recommend a treatment and be done with it. His medical judgment was uncannily accurate. "You mark my words, that man has no more than three days to live," I heard him correctly say once about someone he had observed in a crowd. Perhaps most good physicians develop this prescient skill, but in Juneau, during the 30 years he practiced medicine, he was the best. He also knew when he wasn't. If he didn't have an answer he knew was right he sent his patients to specialists, usually to the place where he began his own professional life, a place he revered all his life: The Virginia Mason Hospital and Clinic in Seattle.

Dr. Whitehead spent his most satisfying professional years delivering babies, a total he estimated at 4,000. "It's the happiest branch of medicine," he said and then added, "I wouldn't be a brain surgeon for all the tea in China." When a committee of U.S. Senators arrived in Juneau during the 1950s to take testimony from Alaskans regarding their expectations of statehood, Dr. Whitehead offered to speak. In a Southern accent made more pronounced by a touch of nervousness, he told the august group: "I spend my professional life delivering babies in this great territory and I would be proud and honored to spend some time with you helping you deliver this territory into statehood."

His patients responded positively and warmly to his folksy approach and bedside manner unless his forthrightness frightened them. He whistled as he walked down the hospital corridors. He liked to visit patients who weren't always his own during his hospital rounds just to say hello and find out what was going on, for he was a social gossip. He always found the right words to make his point during a patient-physician discussion. Take for example, this phone call I remember because it took place at home during our family dinner hour:

Dr. Whitehead: "Tell me, young man, was she
nauseous?
All right, then, did she vomit?
Goddammit, man, did she puke?"

As long as he lived he never lost interest in his profession. Energetic until the day he died from a cerebral hemorrhage while on a deer hunting trip, he took every phone call that came at any hour, made house calls and kept long hours at his office. That kind of life, of course, wasn't good for his health and he paid a terrible price in the end. But he wouldn't have wanted it any other way. He chose to live life vigorously as he understood and wanted to live it.

Bill Whitehead was a complete man. He hoped to have a large family and when that happened he enjoyed the activities of growing children and grandchildren. "It's all very interesting," was as much as he would say critically about some new fad or fancy any of us brought home. He appreciated in his own generational style the beauty, talent and companionship of his wife. "Your mother and I are a team," he would say, noting that he was the team captain. And he considered it a privilege to practice medicine and a privilege to participate in the activities of his community and state as well as in professional and public policy organizations that interested him.

Only once in all those years following his graduation from medical school did he think seriously about returning to Virginia. That was in 1942 when an opportunity came for him to take over the practice of Lynchburg's Dr. A.D.F. White. Our family moved back to Virginia then. But Alaska with its unique opportunities had gotten to him by that time and his Alaskan wife was homesick, so Bill Whitehead returned in 1943 to the "Great Land" so far away from his roots. He and Dorothy, who died in a 1971 automobile accident, are buried side by side in the Alaska Pioneers Plot in Evergreen Cemetery at the foot of Mt. Juneau.

Many fine tributes have been written about our parents over the years but perhaps the finest was inscribed on a plaque that hangs outdoors on the administration and classroom building named in their honor at the University of Alaska Southeast in Juneau.

> **Whitehead Building**
>
> "This first classroom building of the Juneau-Douglas Community College, University of Alaska, is dedicated to the memory of William Massie Whitehead, M.D., 1905-1966, Juneau physician, civic leader, legislator and a member of the University Board of Regents, and to the memory of his wife, Dorothy Johnson Whitehead, 1910-1971, daughter of Alaskan pioneers, mother of five children and a helpmate, always."

Viriginia Whitehead Breeze

Dr. Whitehead liked to deliver things: babies, vegetables, a territory into a state.

This very well might not have been. When he was twelve, he reached down in a potato field to be bitten by a copperhead, a poisonous snake. His father sliced the bite with a razor to make it bleed. Taking a chicken, he cut it open and put Bill's hand into the warm fowl. Then they took the train to Charlottesville, some twenty-five miles away, to the University of Viriginia Hospital. Bill was to spend two weeks in the hospital recovering and all summer with his arm in a sling.

Bill and his brother, Paul, were three years apart in age but close in relationship. They roomed together at the University of Virginia. Both enjoyed a good time. One Sunday they were drinking corn liquor in their room when their father came by, all dressed up for church. One dove under the bed; the other hid in the closet. A resourceful friend opened the door and when asked by their father where they were, replied: "As good students, in the library." Father left.

Bill Whitehead was always gregarious. Especially, he enjoyed the various peoples and cultures he met while shipping with the American Mail Line. Also he bought art work and other mementos which were to be cherished by him lifelong and now by his children.

Dorothy, his wife, had a fascinating mother, born in Norway. As a young woman she had been a reporter for a Minneapolis newspaper, no ordinary thing at the turn of the century for a woman and one whose first language was not English. She was married in 1903. After coming to Alaska, she was to serve on the Wrangell School board, as president of the Territorial Tuberculosis Association and run, unsuccessfully, for mayor of Wrangell.

When Dorothy Johnson married William Whitehead, they honeymooned in Seattle, traveling from Wrangell by the Princess Louise. There is something special about the days when Alaska could be reached only by boat. It provided a way of life quite its own.

Dr. Whitehead had as patients many Alaskans who were to be prominent in Alaska's history. He delivered Susan Bartlett, daughter of the then future Senator "Bob" and Vide Bartlett. Susan's older sister, Doris Ann, accidently sliced off the tip-top of a finger. The piece was left hanging by a thread. Dr. Whitehead sewed it back on and told her mother: "Now, Vide, if this doesn't begin to smell in two or three days, I think it will be all right."

He was especially skillful at removing tonsils. He could do so with relatively little pain or loss of blood. Every two years, when the new Public Health doctors appeared, he would teach them how to do tonsillectomies.

At Christmas time in 1958, he was heard to say: "Bill Egan is not well." This was just before William Egan was to be sworn in as Alaska's first Governor on January 3, 1959. When the new Governor became very ill a few days later, Dr. Whitehead considered this a situation for the Virginia Mason Clinic and Governor Egan was air evacuated to Seattle. Dr. Whitehead also had delivered the Egans' son, Dennis.

He saw patients quickly. Sometimes as many as forty or fifty a day. He kept his nurses running and never wasted time.

He believed that BCG was worth giving to prevent tuberculosis and enthusiastically promoted its use, including his own children.

He was a "Renaissance doctor," feeling that a physician should be involved in a myriad of activities other than medicine.

For Juneau, he was a charter member of the Rotary Club, later serving as president. He was twice president of the Chamber of Commerce. He helped form the Juneau Medical Society and was elected its first president. In 1965, he was named the Outstanding Man for the Greater Juneau area.

For Alaska, he was a member of both the Territorial and State Medical Associations: He was president in 1944 and at the time of statehood in 1958. In 1966, he was honored by ASMA with the A.J. Robbins Award for distinguished service to the community by a physician.

He was elected to the State House, serving from 1963-1965. He was not reelected in part because of his strong views on the right to abortion.

He was president of the Alaska Chapter of the

American Cancer Society and served as a national delegate. In 1966, he was given national recognition for his work in this field in Alaska.

He was appointed chairman of the first Alaska State Judicial Council in 1959. The responsibility of the council was to organize the court system for the new state. Dr. Whitehead felt that the state supreme court should be located in Juneau. However, Buell Nesbitt, who was to serve as the first supreme court chief justice, did not want to live in Juneau. Chief Justice Nesbitt's decision may have proved a good thing for Alaska as it has gown but such are the whims which change things.

In 1961, Dr. Whitehead suffered a small stroke which left him temporarily with a weak hand. But this did not prevent him from continuing his practice or his activities. He was appointed to the University of Alaska Board of Regents, 1965 until his death. His daughter, Virginia Breeze, now serves on the Board of Regents, proudly following her father.

Bill and Dorothy Whitehead were lovely and useful people. In this complex and stressed time, it is refreshing to recreate ever so briefly such an orderly and rewarding world.

Gwynneth Gminder Wilson
ASMA Auxiliary Health Project

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President-Elect's Corner

Dear ASMA Members!

Dr. Huffman has decreed that the President-elect shall write a note in Alaska Medicine, and he will write one for "Heartbeat." So be it!

The main chore for the President-elect is planning for the next convention. The 1990 annual meeting will be in Fairbanks, June 7-9. The theme will have something to do with medical ethics, maybe something medico-legal other than malpractice related topics etc.. Suggestions?

With council approval, I've preempted next year's president-elect and am also working on the 1991 meeting. We plan to hold it in Magadan, USSR. You map fans will locate Magadan just west of the base of the Kamchatka Peninsula, 1900 miles due west of Anchorage. Dr. Bill Townsend has contacted the Minister of Health for the area, Dr. Lebdev, and the Head of Hospitals, Dr. Petrovich. They are enthusiastic about hosting us. This might provide a chance to observe medical practice in the Soviet Union--it is guaranteed to make us happier with our system, even with all its faults. The estimated cost for a charter is $750-$1,000 per person round-trip. You will need a passport, Bill will probably be able to arrange visas painlessly.

Recommendation -- If you plan to go, learn the cyrillic alphabet. Russian is quite phonetic. If you can pronounce the letters, you can read. We share many words. You will be surprised at how much you can glean from a copy of Pravda - we're still referred to as "American Imperialists" - which sounds the same in both languages, just don't forget to roll the "Rs."

Donald R. Rogers, M.D.
President-elect
Alaska State Medical Association

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Hypotension: Excessive hypotension is rare in uncomplicated hypertensive patients treated with VASOTEC alone. Heart failure patients given VASOTEC commonly have some reduction in blood pressure, especially with the first dose, but discontinuation of therapy for continuing symptomatic hypotension usually is not necessary when dosing instructions are followed; caution should be observed when initiating therapy (See DOSAGE AND ADMINISTRATION.) Patients at risk for excessive hypotension, sometimes associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death, include those with the following conditions or characteristics: heart failure, hyponatremia, high-dose diuretic therapy, recent intensive diuresis or increase in diuretic dose, renal dialysis, or severe volume and/or salt depletion of any etiology It may be advisable to eliminate the diuretic (except in heart failure patients), reduce the diuretic dose, or increase salt intake cautiously before initiating therapy with VASOTEC in patients at risk for excessive hypotension who are able to tolerate such adjustments (See PRECAUTIONS, *Drug Interactions* and ADVERSE REACTIONS.) In patients at risk for excessive hypotension, therapy should be started under very close medical supervision and such patients should be followed closely for the first two weeks of treatment and whenever the dose of enalapril and/or diuretic is increased Similar considerations may apply to patients with ischemic heart disease or cardiovascular disease in whom an excessive fall in blood pressure could result in a myocardial infarction or cerebrovascular accident

If excessive hypotension occurs, the patient should be placed in supine position and, if necessary, receive an intravenous infusion of normal saline. A transient hypotensive response is not a contraindication to further doses of VASOTEC, which usually can be given without difficulty once the blood pressure has stabilized If symptomatic hypotension develops, a dose reduction or discontinuation of VASOTEC or concomitant diuretic may be necessary.

Neutropenia/Agranulocytosis: Another ACE inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression, rarely in uncomplicated patients but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease. Available data from clinical trials of enalapril are insufficient to show that enalapril does not cause agranulocytosis at similar rates. Foreign marketing experience has revealed several cases of neutropenia or agranulocytosis in which a causal relationship to enalapril cannot be excluded. Periodic monitoring of white blood cell counts in patients with collagen vascular disease and renal disease should be considered

Precautions: *General: Impaired Renal Function.* As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including VASOTEC, may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death.

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine were observed in 20% of patients. These increases were almost always reversible upon discontinuation of enalapril and/or diuretic therapy In such patients, renal function should be monitored during the first few weeks of therapy

Some patients with hypertension or heart failure with no apparent preexisting renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when VASOTEC has been given concomitantly with a diuretic This is more likely to occur in patients with preexisting renal impairment Dosage reduction and/or discontinuation of the diuretic and/or VASOTEC may be required

Evaluation of patients with hypertension or heart failure should always include assessment of renal function. (See DOSAGE AND ADMINISTRATION.)

Hyperkalemia. Elevated serum potassium (> 5.7 mEq/L) was observed in approximately 1% of hypertensive patients in clinical trials. In most cases these were isolated values which resolved despite continued therapy Hyperkalemia was a cause of discontinuation of therapy in 0.28% of hypertensive patients. In clinical trials in heart failure, hyperkalemia was observed in 3.8% of patients, but was not a cause for discontinuation.

Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with VASOTEC. (See *Drug Interactions.*)

Surgery/Anesthesia: In patients undergoing major surgery or during anesthesia with agents that produce hypotension, enalapril may block angiotensin II formation secondary to compensatory renin release If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion.

Information for Patients.

Angioedema: Angioedema, including laryngeal edema, may occur especially following the first dose of enalapril Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to take no more drug until they have consulted with the prescribing physician

Hypotension: Patients should be cautioned to report lightheadedness especially during the first few days of therapy If actual syncope occurs, the patients should be told to discontinue the drug until they have consulted with the prescribing physician.

All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduction in fluid volume. Other causes of volume depletion such as vomiting or diarrhea may also lead to a fall in blood pressure; patients should be advised to consult with the physician

Hyperkalemia Patients should be told not to use salt substitutes containing potassium without consulting their physician

Neutropenia: Patients should be told to report promptly any indication of infection (e.g., sore throat, fever) which may be a sign of neutropenia

NOTE. As with many other drugs, certain advice to patients being treated with enalapril is warranted This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects

Drug Interactions.

Hypotension: Patients on Diuretic Therapy: Patients on diuretics and especially those in whom diuretic therapy was recently instituted may occasionally experience an excessive reduction of blood pressure after initiation of therapy with enalapril. The possibility of hypotensive effects with enalapril can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with enalapril. If it is necessary to continue the diuretic, provide close medical supervision after the initial dose for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and DOSAGE AND ADMINISTRATION.)

Agents Causing Renin Release: The antihypertensive effect of VASOTEC is augmented by antihypertensive agents that cause renin release (e.g., diuretics).

Other Cardiovascular Agents: VASOTEC has been used concomitantly with beta-adrenergic-blocking agents, methyldopa, nitrates, calcium-blocking agents, hydralazine, prazosin, and digoxin without evidence of clinically significant adverse interactions.

Agents Increasing Serum Potassium. VASOTEC attenuates potassium loss caused by thiazide-type diuretics. Potassium-sparing diuretics (e.g., spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium. Therefore, if concomitant use of these agents is indicated because of demonstrated hypokalemia, they should be used with caution and with frequent monitoring of serum potassium. Potassium-sparing agents should generally not be used in patients with heart failure receiving VASOTEC

Lithium: A few cases of lithium toxicity have been reported in patients receiving concomitant VASOTEC and lithium and were reversible upon discontinuation of both drugs Although a causal relationship has not been established, it is recommended that caution be exercised when lithium is used concomitantly with VASOTEC and serum lithium levels should be monitored frequently

Pregnancy—Category C: There was no fetotoxicity or teratogenicity in rats treated with up to 200 mg/kg/day of enalapril (333 times the maximum human dose). Fetotoxicity, expressed as a decrease in average fetal weight, occurred in rats given 1200 mg/kg/day of enalapril but did not occur when these animals were supplemented with saline Enalapril was not teratogenic in rabbits However, maternal and fetal toxicity occurred in some rabbits at doses of 1 mg/kg/day or more Saline supplementation prevented the maternal and fetal toxicity seen at doses of 3 and 10 mg/kg/day, but not at 30 mg/kg/day (50 times the maximum human dose).

Radioactivity was found to cross the placenta following administration of labeled enalapril to pregnant hamsters

There are no adequate and well-controlled studies of enalapril in pregnant women However, data are available that show enalapril crosses the human placenta Because the risk of fetal toxicity with the use of ACE inhibitors has not been clearly defined, VASOTEC® (Enalapril Maleate, MSD) should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus

Postmarketing experience with all ACE inhibitors thus far suggests the following with regard to pregnancy outcome Inadvertent exposure limited to the first trimester of pregnancy has not been reported to affect fetal outcome adversely Fetal exposure during the second and third trimesters of pregnancy has been associated with fetal and neonatal morbidity and mortality

When ACE inhibitors are used during the later stages of pregnancy, there have been reports of hypotension and decreased renal perfusion in the newborn Oligohydramnios in the mother has also been reported, presumably representing decreased renal function in the fetus. Infants exposed *in utero* to ACE inhibitors should be closely observed for hypotension, oliguria, and hyperkalemia. If oliguria occurs, attention should be directed toward support of blood pressure and renal perfusion with the administration of fluids and pressors as appropriate Problems associated with prematurity such as patent ductus arteriosus have occurred in association with maternal use of ACE inhibitors, but it is not clear whether they are related to ACE inhibition, maternal hypertension, or the underlying prematurity

Nursing Mothers Milk in lactating rats contains radioactivity following administration of ^{14}C enalapril maleate It is not known whether this drug is secreted in human milk Because many drugs are secreted in human milk, caution should be exercised when VASOTEC is given to a nursing mother

Pediatric Use Safety and effectiveness in children have not been established

Adverse Reactions: VASOTEC has been evaluated for safety in more than 10,000 patients, including over 1000 patients treated for one year or more VASOTEC has been found to be generally well tolerated in controlled clinical trials involving 2987 patients

HYPERTENSION: The most frequent clinical adverse experiences in controlled trials were headache (5.2%), dizziness (4.3%), and fatigue (3%)

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in controlled clinical trials were diarrhea (1.4%), nausea (1.4%), rash (1.4%), cough (1.3%), orthostatic effects (1.2%), and asthenia (1.1%)

HEART FAILURE The most frequent clinical adverse experiences in both controlled and uncontrolled trials were: dizziness (79%), hypotension (6.7%), orthostatic effects (2.2%), syncope (2.2%), cough (2.2%), chest pain (2.1%), and diarrhea (2.1%)

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in both controlled and uncontrolled clinical trials were fatigue (1.8%), headache (1.8%), abdominal pain (1.6%), asthenia (1.6%), orthostatic hypotension (1.6%), vertigo (1.6%), angina pectoris (1.5%), nausea (1.3%), vomiting (1.3%), bronchitis (1.3%), dyspnea (1.3%), urinary tract infection (1.3%), rash (1.3%), and myocardial infarction (1.2%)

Other serious clinical adverse experiences occurring since the drug was marketed or adverse experiences occurring in 0.5% to 1% of patients with hypertension or heart failure in clinical trials in order of decreasing severity within each category

Cardiovascular: Cardiac arrest, myocardial infarction or cerebrovascular accident, possibly secondary to excessive hypotension in high-risk patients (see WARNINGS, *Hypotension*), cardiac arrest, pulmonary embolism and infarction, rhythm disturbances, atrial fibrillation, palpitation

Digestive Ileus, pancreatitis, hepatitis or cholestatic jaundice, melena, anorexia, dyspepsia, constipation, glossitis

Nervous/Psychiatric. Depression, confusion, ataxia, somnolence, insomnia, nervousness, paresthesia

Urogenital Renal failure, oliguria, renal dysfunction (see PRECAUTIONS and DOSAGE AND ADMINISTRATION).

Respiratory Bronchospasm, rhinorrhea, asthma, upper respiratory infection

Skin. Herpes zoster, pruritus, alopecia, flushing, photosensitivity

Other. Vasculitis, muscle cramps, hyperhidrosis, impotence, blurred vision, taste alteration, tinnitus

A symptom complex has been reported which may include fever, myalgia, and arthralgia, an elevated erythrocyte sedimentation rate may be present Rash or other dermatologic manifestations may occur. These symptoms have disappeared after discontinuation of therapy

Angioedema Angioedema has been reported in patients receiving VASOTEC (0.2%) Angioedema associated with laryngeal edema may be fatal If angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with VASOTEC should be discontinued and appropriate therapy instituted immediately (See WARNINGS.)

Hypotension: In the hypertensive patients, hypotension occurred in 0.9% and syncope occurred in 0.5% of patients following the initial dose or during extended therapy Hypotension or syncope was a cause for discontinuation of therapy in 0.1% of hypertensive patients In heart failure patients, hypotension occurred in 6.7% and syncope occurred in 2.2% of patients Hypotension or syncope was a cause for discontinuation of therapy in 1.9% of patients with heart failure (See WARNINGS.)

Clinical Laboratory Test Findings

Serum Electrolytes Hyperkalemia (see PRECAUTIONS), hyponatremia

Creatinine, Blood Urea Nitrogen. In controlled clinical trials, minor increases in blood urea nitrogen and serum creatinine, reversible upon discontinuation of therapy, were observed in about 0.2% of patients with essential hypertension treated with VASOTEC alone Increases are more likely to occur in patients receiving concomitant diuretics or in patients with renal artery stenosis (See PRECAUTIONS.) In patients with heart failure who were also receiving diuretics with or without digitalis, increases in blood urea nitrogen or serum creatinine, usually reversible upon discontinuation of VASOTEC and/or other concomitant diuretic therapy, were observed in about 11% of patients Increases in blood urea nitrogen or creatinine were a cause for discontinuation in 1.2% of patients

Hemoglobin and Hematocrit. Small decreases in hemoglobin and hematocrit (mean decreases of approximately 0.3 g % and 1.0 vol %, respectively) occur frequently in either hypertension or heart failure patients treated with VASOTEC but are rarely of clinical importance unless another cause of anemia coexists In clinical trials, less than 0.1% of patients discontinued therapy due to anemia

Other (Causal Relationship Unknown) In marketing experience, rare cases of neutropenia, thrombocytopenia, and bone marrow depression have been reported

Liver Function Tests Elevations of liver enzymes and/or serum bilirubin have occurred

Dosage and Administration: *Hypertension* In patients who are currently being treated with a diuretic, symptomatic hypotension occasionally may occur following the initial dose of VASOTEC The diuretic should, if possible, be discontinued for two to three days before beginning therapy with VASOTEC to reduce the likelihood of hypotension (See WARNINGS.) If the patient's blood pressure is not controlled with VASOTEC alone, diuretic therapy may be resumed

If the diuretic cannot be discontinued, an initial dose of 2.5 mg should be used under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions.*)

The recommended initial dose in patients not on diuretics is 5 mg once a day Dosage should be adjusted according to blood pressure response The usual dosage range is 10 to 40 mg per day administered in a single dose or in two divided doses. In some patients treated once daily, the antihypertensive effect may diminish toward the end of the dosing interval In such patients, an increase in dosage or twice-daily administration should be considered If blood pressure is not controlled with VASOTEC alone, a diuretic may be added

Concomitant administration of VASOTEC with potassium supplements, potassium salt substitutes, or potassium-sparing diuretics may lead to increases of serum potassium (see PRECAUTIONS)

Dosage Adjustment in Hypertensive Patients with Renal Impairment. The usual dose of enalapril is recommended for patients with a creatinine clearance >30 mL/min (serum creatinine of up to approximately 3 mg/dL) For patients with creatinine clearance ≤30 mL/min (serum creatinine ≥3 mg/dL), the first dose is 2.5 mg once daily The dosage may be titrated upward until blood pressure is controlled or to a maximum of 40 mg daily

Heart Failure: VASOTEC is indicated as adjunctive therapy with diuretics and digitalis. The recommended starting dose is 2.5 mg once or twice daily After the initial dose of VASOTEC, the patient should be observed under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour (See WARNINGS and PRECAUTIONS, *Drug Interactions.*) If possible, the dose of the diuretic should be reduced, which may diminish the likelihood of hypotension The appearance of hypotension after the initial dose of VASOTEC does not preclude subsequent careful dose titration with the drug, following effective management of the hypotension The usual therapeutic dosing range for the treatment of heart failure is 5 to 20 mg daily given in two divided doses The maximum daily dose is 40 mg Once-daily dosing has been effective in a controlled study, but nearly all patients in this study were given 40 mg, the maximum recommended daily dose, and there has been much more experience with twice-daily dosing In addition, in a placebo-controlled study which demonstrated reduced mortality in patients with severe heart failure (NYHA Class IV), patients were treated with 2.5 to 40 mg per day of VASOTEC, almost always administered in two divided doses (See CLINICAL PHARMACOLOGY, *Pharmacodynamics* and *Clinical Effects.*) Dosage may be adjusted depending upon clinical or hemodynamic response. (See WARNINGS.)

Dosage Adjustment in Heart Failure Patients with Renal Impairment or Hyponatremia In heart failure patients with hyponatremia (serum sodium <130 mEq/L) or with serum creatinine >1.6 mg/dL, therapy should be initiated at 2.5 mg daily under close medical supervision. (See DOSAGE AND ADMINISTRATION, *Heart Failure*, WARNINGS, and PRECAUTIONS, *Drug Interactions.*) The dose may be increased to 2.5 mg b.i.d., then 5 mg b.i.d. and higher as needed, usually at intervals of four days or more, if at the time of dosage adjustment there is not excessive hypotension or significant deterioration of renal function. The maximum daily dose is 40 mg

For more detailed information, consult your MSD Representative or see Prescribing Information Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, PA 19486

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GLIMPSES OF ALASKAN MEDICAL HISTORY

Edited by Robert Fortuine, M.D.

IN STYLE ON KODIAK ISLAND (1804-7)

Father Gedeon, cathedral hieromonk at the Aleksandrs-Nevskii monastery near St. Petersburg, arrived in Kodiak in July 1804 and remained on the island until June 1807. He had been sent to the distant colonies by the Holy Synod in response to a collective complaint by members of the Russian Orthodox mission against Chief Manager Aleksandr Baranov and other officials of the Russian-American Company. Gedeon's orders were to "look into the situation of the newly baptized American [i.e. Native] Christians and of the missionary establishment there." During his three-year stay he mediated the quarrels between the churchmen and the distinctly secular members of the trading establishment.

While at Kodiak the hieromonk tried to lift the spirits of the now demoralized missionaries. He reorganized the school and for the first time held public examinations for the students. He also became one of many before and after who attempted to develop agriculture on the island. Foreshadowing the great Father Veniaminov a generation later, Father Gedeon made the first efforts toward compiling an Aleut dictionary. On his departure from the island he took with him a protégéCreole (mixed-blood) student, who some time later returned to Kodiak as an Orthodox priest.

Father Gedeon was an exceptionally learned man, with a background in logic, rhetoric, geography, history, mathematics, physics, French, and philosophy, as well as theology. Upon his return to Russia in 1808 he submitted a detailed report of his activities, together with his correspondence with high officials, including Baranov and Nikolai Rezanov. He also prepared detailed notes on the Russian colony at Kodiak, and, perhaps even more valuable, a treatise on nearly every aspect of Koniag culture, including healing.

The text below, from his ethnographic notes, is important medically because it demonstrates the types of surgical procedures on their lips, noses, ears, or face that nearly all Koniag had to undergo at some period of their lives. Similar records could be cited for nearly every Alaska Native culture.

"The Kadiak Americans are of medium height, unusually athletic in build, but for the most part round-shouldered. Their faces are somewhat rounded, swarty--though some are pale and ruddy-cheeked. They have a medium-sized nose; their eyes and hair are black, with thick eyebrows, broad ears, their lips somewhat thick and their teeth white. Both men and women in almost the whole surrounding area have their ears pierced and wear tsukli or beads there, or the mother-of-pearl referred to above, or pieces of coral, and the rich will wear two pieces of amber. The older men have slit lower lips and an incision made between the nostrils. Formerly every woman, without exception, had a tattooed chin, and some still have tattooed on their body two broad stripes criss-crossing both shoulders with various patterns--this is thought to be a sign of the height of fashion. Such patterns are also worn by some of the men. This tattooing is achieved by lifting a flap of skin with a needle so that a little blood flows. This is then rubbed with ink made from the ash of the fir tree and mixed with the blood; this then produces a dark blue color against the skin. The women have in their lower lip five to six small holes from each of which hangs a bead attached to a thread some half a chetvert long. And in the hole pierced in their nostrils they wear the thin twisted tooth of some animal. Their ears are weighed down with beads.... On their necks, arms and legs they wear bracelets made of various colored beads some vershoks in width, and others wear on their ankles or wrists some five to six rings made of iron or brass. At the festivals, especially when the tolkushka is being served, and at other times of celebration they decorate or simply smear their faces with red or some other color pigment--apart from black, which is a sign of mourning. The men too paint their faces on such occasions, especially when they are approaching their villages as they return from the hunt."

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My Turn

Dr. Jack of Calcutta

May, 1988. The Fairlawn Hotel on Sudder Street, just off the busy Chowringhee, was British Raj-era oasis, a haven from the bustling, teeming masses of Calcutta. The ceiling fans even provided some relief from the heat, but there was no refuge anywhere from the all-pervasive humidity. It attacked my paper-back, curling the pages until they stood at right angles. A bar of soap left on the sink turned to mush. Clothes that I had washed and set out to dry were still wet after two days. The coating on my nightstand, absorbed so much moisture that it melted into a small pink puddle. It was another hot and sticky morning when I set out with Mohammed, and Algerian journalist from London, to look for Dr. Jack.

I'd been hearing bits and pieces about Dr. Jack since I arrived in Calcutta. He was a British physician who had conducted a streetside clinic for the poor of Calcutta for about eight years. He also provided medical care to the many low-budget foreigners passing through. People we met on the street spoke of him with great respect. He was the subject of a documentary at the Cannes Film Festival and also was said to be a candidate for the next Nobel Peace Prize. But he was also in serious trouble with the Indian government.

We arrived at Middleton Row about midmorning. His patients were already beginning to gather, squatting Indian-style along the sidewalk. Cripples, asthmatics, people with tuberculosis, gastrointestinal bleeding, skin wounds. Mothers clutched sick infants. Dr. Jack's total patient population was in excess of 6,000, which probably explains some of the Indian government's increasing uneasiness with him.

A skinny Australian resident presided over a group of volunteers, fumbling through boxes of dog-eared medical records and jars of medications that had survived previous police raids.

Dr. Jack arrived. Dressed in soiled white pants and a plaid shirt clinging with sweat, he seemed a very unassuming individual. A Canadian journalist introduced me to him, and we chatted briefly. Then he spoke for a few minutes with the Australian resident before disappearing into the back seat of a taxi, off to an Indian courtroom to fight deportation.

Epilogue:

Dr. Jack had mentioned to me that his brother was a radiologist in San Francisco. I was able to get his phone number through the Alaska Health Sciences Library and gave him a call, asking for an update on his brother in Calcutta. I was relieved to hear that Dr. Jack was still hard at work treating the poor on the streets of Calcutta. With the backing of the British Medical Association he has apparently been able to successfully fight off deportation. Hopefully, he will be able to do so indefinitely.

William M. Cox, M.D.
February, 1989

Auxiliary News

Alaska State Medical Association Auxiliary

The primary role of the State Auxiliary is to assist the State Medical Association in programs that improve the health and quality of life for all people; to promote health education; encourage volunteers in activities that meet health needs; and to support health related, charitable endeavors.

Though the Alaska State Auxiliary consists of one local society, Anchorage, and a few members at-large from other communities, we hope to expand our membership this year through new society auxiliaries in other areas of Alaska. Most health related issues are initially addressed on a local level, and with one active society we cannot be as effective throughout the state as we would like to be. It is the State Auxiliary's goal for 1989-90 to add at least one other society auxiliary.

Lindsay Hansen Tucker
President, ASMA Auxiliary

Anchorage Medical Society Auxiliary

As the president of the Anchorage Medical Society Auxiliary for the year 1989/90, please allow me to introduce myself. My name is Tammy Horton. I am the wife of Christopher Horton, M.D., orthopaedic surgeon, mother of one 3-year-old and pregnant, (due November 15), Vice President of The Private Cable Company, President of Pacific Rim Telecommunications, and yes, I do have time for the auxiliary.

I know that you and your spouse are busy, however I invite you to encourage your spouse to join and participate with the auxiliary, because I believe we, as a medical community have a responsibility to ourselves to be united in as many ways as possible, to keep the things we like and change those we do not. Together, the medical community can do a lot for ourselves that will benefit the community as a whole.

A Holiday Ball is planned for this Christmas Season. Please mark your calendar.

Date:	Saturday, December 16, 1989
Place:	Sheraton Anchorage Howard Rock Ballroom
Cost:	$60.00 per couple
Dress:	Black Tie optional

Please look for the invitations due to be mailed in November. The Ball is being sponsored by the Anchorage Medical Society and Auxiliary, Alaska Native Medical Center, Elmendorf Air Force Base Hospital, Humana Hospital - Alaska, and Providence Hospital. We look forward to seeing you there.

Thank you for your time. Please call me if I can be of any assistance to you or if you have any suggestions.

Tammy J. Horton
President, AMS Auxiliary

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Guest Editorial

A REFUGE FROM IMPOSSIBLE EXPECTATIONS

Even though they promise us excellent work, we don't persecute good carpenters who hang an occasional door backwards. We ask them to pay for the replacement and hang it again. We don't impeach the politicians who include loopholes in even good laws. We don't blackball stockbrokers who give us one bum suggestion along with the good ones. And yet, the public repudiates physicians who cannot bat 1.000. And what's worse, we physicians agree with these impossible expectations by deluding ourselves that we are different, and can extract continuous perfect performance from ourselves.

Biologically we are made of the same material that carpenters, politicians and stockbrokers are. Our span of concentration is prone to the same lapses. We can be overconfident or distracted. We can mishear, misunderstand, misread. We can be too proud to admit we don't know or understand. We can be dulled by alcohol, drugs, or other compulsions. Money troubles or similar "outside" influences can pressure us to compromise our own standards. We can be too tired to make a good decision, or too emotionally involved to see the situation fairly.

The standards set for us are higher than for other professions, but we are made of the same fallible stuff. In fact, the harder we try to be perfect, the more we increase the probability of certain types of failure. Excessive internal pressure pushes us to chemicals for relaxation; the zeal not to miss a possibility results in exorbitant charges, and so on.

Lawyers, insurers, and the public refuse to accept the inevitability of our frailties. We are exhorted, goaded, expected and even ordered to meet "minimum standards." And if we claim that peak performance at every moment cannot be a "minimum" standard, we are looked at with suspicion. Why are we unwilling to assure all patients such a "simple" thing as this reasonable "minimum" of care? Who can disagree that each patient deserves a caring and competent and attentive physician at all times. But where can we find human beings who can be that way 100% of the time?

Other areas of our culture have zero tolerance for error, like the Defense Department and NASA. Those areas now use duplicate and triplicate systems, with fail-safe overrides. Medical decision-making does not fit that model. The medical culture has reacted to the need for unerring performance by expecting more of the same basic equipment. We merely tighten up our rear ends, and look behind our shoulder all the time.

Constantly on guard against mistakes, we feel surrounded by dangers:

- the danger of errors of omission, forgetting something;
- the danger of errors of commission, doing something incorrectly;
- the danger of careless speech, of admitting error, vulnerability, uncertainty or guilt;
- the danger of attack, by vengeful or crazy patients, predatory lawyers, or merciless automaton bureaucrats;
- the danger of one moment of inattention, that hurts someone, and that eventually wipes out EVERYTHING.

One moment of inattention could destroy our reputation, our livelihood, our social standing and material security, our own sense of competence, our self-worth and "peace of mind." It can invalidate a lifetime of sincere effort and good work.

Society's loss of respect for physicians weighs on us heavily. We feel used as scapegoats for the inescapable raw deals of life--pain, loss and death. Our motives are suspect, with our commitment to money overestimated and our commitment to healing underestimated. Those who feel vulnerable, powerless and dependent on us resent us. Those who think we are rich, arrogant and immune from the inconveniences of everyday life look for opportunities to bring us low.

And here's the sad part: We do not support one another and admit the impossibility of society's expectations. We rail against lawyers, bureaucrats and society, but we do not give comfort and protection to each other. Instead, we keep so busy on the perfection treadmill, that we have no time to spend with those who are in trouble.

Few physicians say they are happy to be in medicine these days. Most seem to be overcommitted and drained of energy. How can we keep our courage up, be proud of our choice of an honorable and fulfilling profession, and find ways to work around our natural inability to be perfect?

In my opinion, the Alaska State Medical Association, and its component local societies have an obvious but neglected role to play in tending to the emotional tension and spiritual near-exhaustion of the state's physicians. The major growth in ASMA in the last few years has been focussed outward--developing ties with the legislature, the bureaucracy, and the public. That work is essential and will continue. But perhaps we need to focus new efforts inward as well. By inward, I mean more than traditional member services like directories, newsletters and informational meetings.

By inward, I mean that ASMA could serve as solace for us, and provide a place and people we can come to for acceptance, help, and a sympathetic ear. Our Impaired Physicians Program has had a good start, but it is aimed at the small group that has become clinically ill. Some other states have formed malpractice support groups, acknowledging that a law suit may be as much or more of a crisis as divorce or death. At least one state society has a group that meets monthly for mutual support at breakfast. The ASMA Council meeting in October will hear more about this new "therapeutic" role for ASMA. The Anchorage Medical Society meeting in February will feature a medical ethicist who will help us talk about our human frailty, and the pressures for perfection. If these beginnings are successful, more events will be planned.

Those of us who are active in organized medicine in Alaska are seeking ways to serve our constituents--Alaskan physicians--better. In order to improve our healing of others, perhaps we can begin by healing ourselves.

Jennifer Christian, M.D.
Municipality of Anchorage
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ALASKA MEDICINE

Official Journal of:

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Volume 32, Number 1 Jan/March 1990

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Volume 32 January/February/March Number 1

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Chukchi reindeer herder and son, north of Bilibino City, Magadan Region, USSR. Photo courtesy of Theodore Mala, M.D., M.P.H., Director, Institute for Circumpolar Health Studies, University of Alaska Anchorage.

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Respiratory Syncytial Virus and Ribavirin Therapy:
Winter 1987 - Spring 1988 A Report of 74 cases treated at Providence Hospital, Anchorage, Alaska

by Jon Lyon, M.D.(1)
Dion Roberts, M.D.(1)
David Brauner, D.O.(1)
Jody Harris, R.N., M.N.(1)
Dawn Pope, R.N., B.S.(1)

ABSTRACT

Seventy-four pediatric cases of Respiratory Syncytial Virus lower respiratory tract infection were treated at Providence Hospital, Anchorage, Alaska between September 1, 1987 and April 30, 1988. These patients are described with illustrative case reports. Twenty-one patients were treated with Ribavirin and these patients are further analyzed as a special interest group. A disproportionate number of the Ribavirin treated group were Alaskan Natives. In the discussion we address concerns of possible toxicity of Ribavirin and current recommendations for patient selection for treatment with Ribavirin.

INTRODUCTION

During the winter of 1987-1988 Alaska experienced a major outbreak of Respiratory Syncytial Virus (RSV) lower respiratory tract infections in infants and children. The epidemic received statewide attention among physicians and the lay community. We wish to report briefly the experience of the Pediatric Service at Providence Hospital, Anchorage, with infants and children. Patients were admitted from the Anchorage area or referred from outlying areas. This review has been made timely by the recent availability of rapid diagnostic techniques for RSV and pharmacologic treatment with ribavirin, an antiviral agent.

CASE REVIEWS

We reviewed cases between September 1, 1987 and April 30, 1988 of infants and children diagnosed suffering from RSV lower respiratory tract infections by Enzyme Immunoassay (EIA) of nasal washings. The records of all patients treated with Ribavirin were further analyzed as a special interest group.

ILLUSTRATIVE CASE REPORTS

Case 1

A 7-month old Alaska Native infant was admitted to Providence Hospital for elective abdominal surgery. The initial physical examination revealed crusting about the nares, but did not otherwise indicate respiratory disease. The preoperative period was uneventful, however, during anesthesia copious airway secretions were encountered. When intermittent mild hypoxemia occurred, surgery was cancelled. Repeat examination that day revealed otitis media. Chest x-rays revealed hyperinflation of the lungs, but were otherwise unchanged from previous studies. The day following anesthesia expiratory wheezing occurred. Nasal washing was positive for RSV by EIA. Two days later the infant was free of symptoms and taking an antibiotic for otitis media at discharge.

COMMENT: Case 1 is typical of the majority of infants without predisposing causes of severe disease who recovered uneventfully without intervention, and might not have come to medical attention under other circumstances.

Case 2

A 13-year old Anchorage boy with known cardiomyopathy was transported to Providence Hospital by ambulance complaining of right chest pain, cough, and increasing shortness of breath. Chest examination revealed generalized rales and x-rays showed perihilar infiltrates. Oxygen administration was associated with abatement of the chest pain. Sputum culture yielded group B streptococcus and a nasal washing was positive for RSV by EIA. Ribavirin therapy was given for three days. He was asymptomatic by the end of the ribavirin course and was discharged two days later receiving penicillin and his usual cardiac medications.

---

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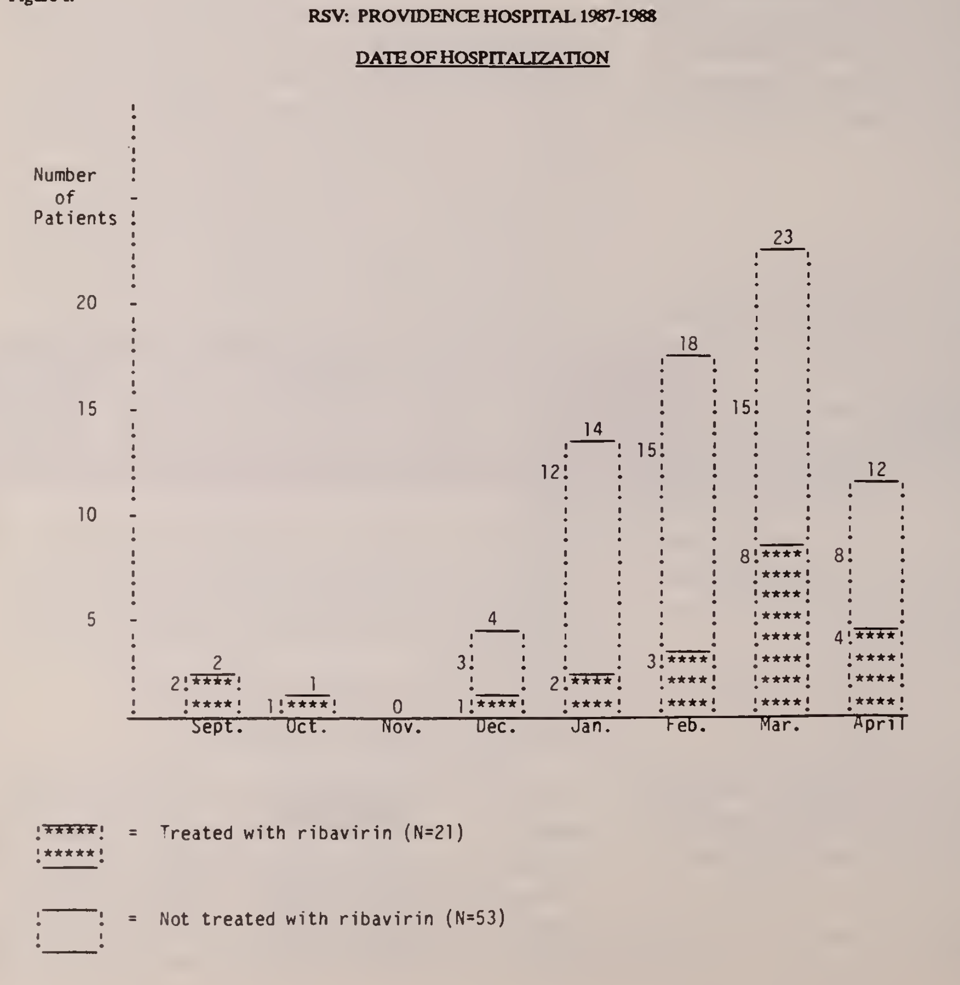
COMMENT: Although RSV infection is generally a benign condition in children beyond the first one to two years of life, serious RSV illness may occur in older individuals already compromised by cardiac or pulmonary disease.

Case 3

A 6-week old male Alaska Native developed a "cold" with sneezing, rhinitis, and cough. His symptoms increased over a five day period. Recurrent apnea developed the night before admission to USPHS Hospital, Bethel. Initial pulmonary examination revealed bilateral coarse crackles. Chest x-rays demonstrated right upper lobe and lingular infiltrates. Recurrent apnea persisted despite administration of 100% oxygen. Ceftriaxone was administered, tracheal intubation performed, and mechanical ventilation initiated. Seizures were noted during transfer to Providence Hospital. A nasal washing was positive for RSV by EIA, and RSV was subsequently cultured. WBC numbered 9400/mm3 with the differential showing predominantly neutrophils and precursors. Hct was 34.7% with hemoglobin 11.7 gm/dl. Total serum protein was 4.5 gm/dl with albumin 2.6 gm/dl. Treatment after transfer consisted of intravenous hydrocortisone, nebulized metaproterenol, phenobarbital, Dilantin, and ribavirin aersol. Right upper lobe atelectasis complicated the infant's course, but he was extubated after three days and oxygen was discontinued after nine days.

COMMENT: Case 3 illustrates the uncertainties that may be encountered when treating RSV infections. The precarious condition of the infant compelled the use of ribavirin during mechanical ventilation, a technique for which appropriate dosage has not been established and one that introduces a risk of ventilator malfunction.

Figure 1.



RESULTS

During the study period, (Figure 1) 74 individuals hospitalized at Providence Hospital, ranging in age from 2 weeks to 13 years (Figure 2), were diagnosed as having RSV lower respiratory tract disease by a positive EIA performed at Humana Hospital Alaska laboratory. Of these, 21 patients were treated with ribavirin. In this subgroup, EIA performed by the Alaska State Laboratory was in agreement for four of five cases in which specimens were submitted to both institutions. In addition, ten infants had viral cultures: five were negative, three grew RSV, one grew rhinovirus, and one grew cytomegalovirus. Nine infants required mechanical ventilation. There were three deaths, two in the Ribavirin-treated group.

The two deaths in the treated group were infants with severe RSV infection and respiratory failure who, in addition, had significant underlying illness; in the first case, recurrent aspiration syndrome, and in the second, prematurity with siezure disorder. The death in the untreated group was a child with less severe RSV infection, but with cerebral palsy with siezure disorder and hydrocephalus, who died of a complication of a ventriculo-peritoneal shunt.

The length of stay at Providence Hospital ranged from 5 to 32 days. The mean length of stay for the entire study group was five days. For the group not treated with Ribavirin (N=53) the mean was four days; for the Ribavirin-treated group (N=21) the mean was ten days

The duration of Ribavirin therapy (Figure 3) was predominantly 3-5 days. Two infants treated for less than two days had ribavirin therapy discontinued when their deterioration was thought to be related to the medication.

Other demographic data (Table I) suggest a disproportionate morbidity among Alaskan Native infants. In addition to classic lower respiratory disease, five infants from the Yukon-Kuskokwim area had seizures as major presenting symptoms. The indicators for Ribavirin treatment (Table II) are derived from the American Academy of Pediatrics Policy Statement regarding candidates for treatment with Ribavirin (see Discussion). The most common indicator for Ribavirin treatment other than young age was respiratory failure. The most common underlying pulmonary disease was bronchopulmonary dysplasia in premature infants.

Table I.

	RIBAVIRIN	
	TREATED (N= 21)	NOT TREATED (N = 53)
RACE		
White	9 (43%)	37 (70%)
Black	1 (4%)	3 (6%)
Alaskan Native	11 (53%)	11 (20%)
Other	0 (0%)	2 (4%)
GEOGRAPHIC ORIGIN		
Anchorage	9 (42%)	47 (88%)
Yukon/Kuskokwin/ Bristol Bay	10 (47%)	6 (12%)
Kodiak	1 (4%)	0 (0%)
Kenai Peninsula	1 (4%)	0 (0%)
SEX		
Male	9 (43%)	36 (67%)
Female	12 (57%)	17 (33%)

Figure 2.

RSV: PROVIDENCE HOSPITAL 1987-1088

AGE OF STUDY PATIENTS

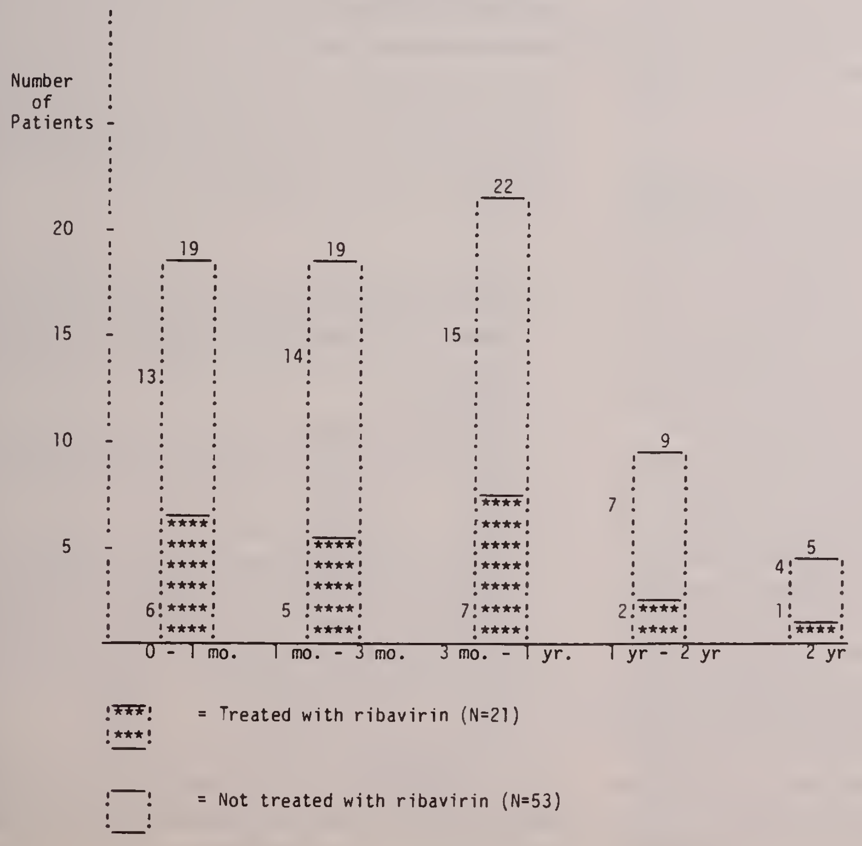


Table II.

INDICATORS FOR TREATMENT IN RIBAVIRIN TREATED AND NOT TREATED PATIENTS

	RIBAVIRIN	
	TREATED (N = 21)	NOT TREATED (N = 53)
INDICATORS FOR TREATMENT WITH RIBAVIRIN*		
age less than 3 months	11	27
cardiac	3	2
pulmonary	5	12
respiratory failure	10	0
neurological	6	3

*may be more than one for each patient

DISCUSSION

In this paper we describe our experience with a winter outbreak of RSV lower respiratory tract infection. This report is not meant to be an epidemiologic report and we recognize that new cases continued to occur after our sampling time period. We have not attempted to describe the patients with lower respiratory tract infections in our hospital who had negative RSV EIA during this time period nor to seek out among lower respiratory tract infections undiscovered RSV cases.

We based the diagnosis of RSV disease on a positive RSV EIA nasal washing performed in a local laboratory. This test is felt to be accurate with a 73%-93% sensitivity and a 92%-99% specificity (1,2). As is demonstrated here and in other studies (3), it is possible to have other concurrent and complicating infections which may contribute excessive morbidity. It is wise, therefore, particularly in the most ill individuals to obtain other cultures, both bacterial and viral.

Ribavirin is a synthetic nucleoside analog resembling guanasine and inosine that appears to interfere with the expression of messenger RNA and to inhibit viral protein synthesis while not being significantly incorporated into host RNA or DNA. Ribavirin can be administered as an aerosol with particles small enough to reach the lower respiratory tract via oxygen hood or tent. It can also be administered via ventilator; however, use of Ribavirin in ventilator-dependent individuals presents unique difficulties in management. The material exhibits a strong tendency to accumulate in the circuits and valves of ventilator systems with the potential for disastrous malfunctions. A recent publication has described modifications to obviate this difficulty (4). Thus far, however, appropriate dosage has not been established for administration through a ventilator. Anecdotal reports generally attribute adverse reactions to reactive airway disease or pulmonary edema. Physiologic basis for both exist.

As Ribavirin became available in late 1986, the American Academy of Pediatrics released a policy statement on Ribavirin treatment of RSV, reviewing concerns about the possible toxicity of Ribavirin including studies in which teratenogenicity has been observed in treating pregnant rodents, and where concerns are raised regarding the molecular similarity of Ribavirin to oncogenic drugs (5). In addition, incidental exposure of personnel and visitors to Ribavirin aerosol is likely to occur. In reported studies exposures to personnel were greatest when Ribavirin was administered by oxygen tent, less by mist tent, and least by ventilator (6). The Centers for Disease Control recommend that until a specifically designed control system is developed to reduce aerosol emissions, health care workers and visitors who are pregnant or may become pregnant should be advised of the potential risks of exposure when patients are receiving Ribavirin and should be counseled about risk reduction strategies (6).

Though its efficacy has not been conclusively demonstrated, and is being challenged, it is currently held that in high risk individuals, and for those with severe respiratory compromise, Ribavirin is beneficial. However, until risks associated with Ribavirin use are elucidated, ribavirin should not be used indiscriminately and patients should be carefully selected (7,8).

The Academy Policy Statement (5) recommends that candidates for treatment with Ribavirin be infants hospitalized with lower respiratory tract disease caused by RSV in one of three categories:

1) Infants at high risk for severe or complicated RSV infections; including infants with congenital heart disease, bronchopulmonary dysplasia, other chronic serious lung conditions, or children with immunodeficiency.
2) Severely ill infants with evidence of hypoxia (Pa02<65%) or increasing hypercarbia, or acidosis.
3) Infants who are not initially severely ill, but who may be at some increased risk of progressing to more complicated course, e.g., infants less than six weeks of age, infants with an underlying condition such as multiple congenital anomalies, neurologic, or metabolic diseases.

All of our patients treated with Ribavirin met at least one of these criteria, often more than one. Simply falling into one of these categoreies, however, does not automatically indicate that Ribavirin be used; clinical judgement regarding the severity of the manifestations of the criteria in individual patients is warranted.

The RSV epidemic in Alaska received much attention from the press and was worrisome to Alaska parents and health professionals alike. The CDC is conducting an epidemiologic study of the outbreak and more information should be available in the future. RSV though, is the most important cause of lower respiratory tract disease in infants and young children. We can expect it back each year. Most previously healthy infants who acquire RSV and require hospitalization will do well with only supportive care. For the more severely ill infant with underlying high risk factors, care in a specialized pediatric center and consideration of the use of ribavirin are indicated.

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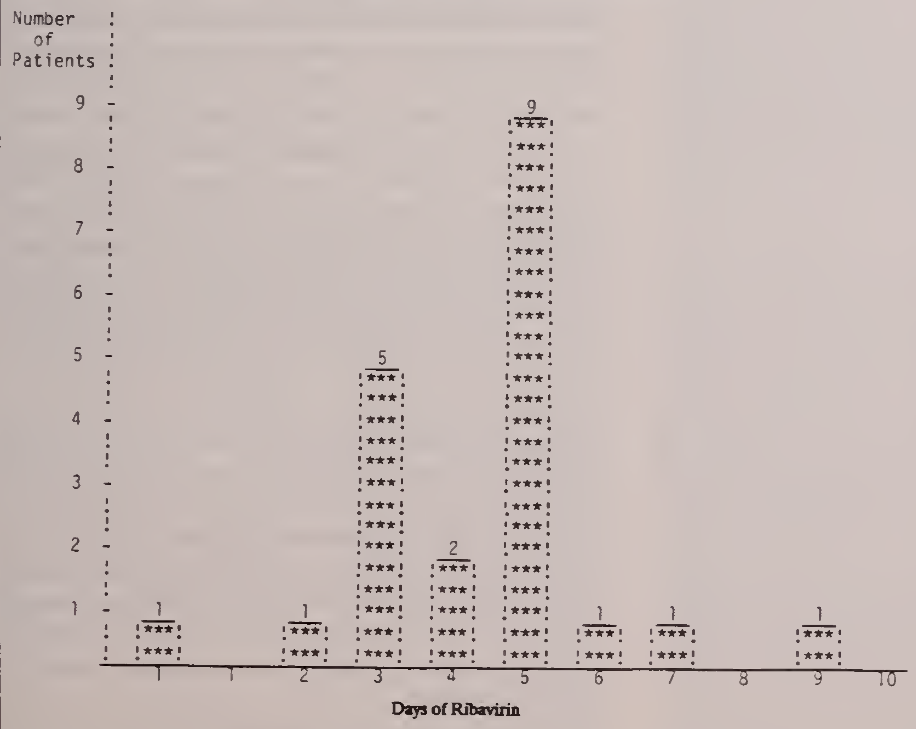
Figure 3.

RSV: PROVIDENCE HOSPITAL 1987-1988

DURATION OF RIBAVIRIN TREATMENT



Mat-Su Borough EMS

by Kevin Koechlein(1)
Mark S. Johnson(2)

INTRODUCTION

The Matanuska-Susitna (Mat-Su) Borough encompasses over 23,000 square miles, with thirteen small and midsize rural communities ranging in size from Gold Creek (population 36) to Wasilla (population 3,977). Only Palmer (population 3,116) has a hospital with a total of 40 beds. In 1986, the total population of the Borough was estimated to be 44,280 by the Alaska Department of Community and Regional Affairs and the Borough.

Like many other rural highway areas in southcentral and interior Alaska, the Mat-Su Borough has long stretches of highways between communities, including the George Parks and the Glenn Highways.

HISTORICAL BACKGROUND

Prior to 1976, EMS in the Mat-Su Borough was provided by ambulance services located in Palmer and Wasilla. At that time the only training available for the volunteers was advanced first aid. Attempting to cover the entire Borough road system often resulted in very long response and transport times. Beginning in early 1975, with the assistance of the Regional EMS Council, a number of concerned volunteers met to formulate a plan for a Borough-wide system of ambulances and trained volunteers. This core group became the first Borough EMS Advisory Board and was made up primarily of people from all parts of the Borough and Valley Hospital in Palmer.

In their planning discussions it was determined that a Borough-wide EMS system was preferable to a system based on service areas and limited response capability. This initial group also began the strong commitment to training and equipping the volunteers in order for them to adequately perform their service to the community. The Advisory Board was able to gather the necessary signatures to place the issue on the ballot in October 1976. The proposition passed overwhelmingly and the Mat-Su EMS Program was born.

Within the next few months, the Advisory Board formulated an operating budget and began training volunteers in Willow, Talkeetna, Wasilla, and Palmer. Through a series of State grants, ambulances, and equipment were acquired and placed in service. By summer of 1977 the program was off and running. Many of the people involved in this early stage are still involved in EMS or other community activities that support the Mat-Su Borough EMS program.

As the Borough continued to grow, new service locations were added as funding became available. One reason the Borough EMS program has been able to respond to population growth is the long-standing commitment by the Borough Assembly to use local tax dollars to fund the program. This has created a stable funding base that gives the program stability and flexibility. By 1980 ambulances were stationed in eight locations and the Board was working on the first advanced level EMT training program to improve its services. With the initiation of EMT-II and, in 1982, EMT-III level care, it was felt that a more permanent form of management was needed. This was accomplished in 1982 with the hiring of a full time EMS Coordinator to direct and administer the EMS program.

Combining the talents of a dedicated group of volunteers with a progressive medical director and full time manager allowed the EMS program to begin a dramatic growth period. Within two years every ambulance service was at the EMT-II level and three were at the EMT-III level. Organized first responder programs were initiated in many areas to further reduce response times and improve the delivery of field care. Support programs were begun to deal with extrication and water rescue. Special programs aimed at assisting the volunteers with their needs also were initiated, such as Critical Incident Stress Debriefing for emergency responders, health screening and a physical fitness program.

When one looks at the response today in the Borough to a vehicle accident, it becomes immediately apparent that the system has come of age. From the beginning in 1976 of having a few advanced first aid trained volunteers trying to cover the entire Borough, it has evolved into a complex system of local ambulance stations staffed with advanced level EMTs and Rescue Technicians backed up with a strong management system and support program.

This rapid advancement has placed the Mat-Su Borough in the forefront of both technology in EMS

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systems as well as in training and educational opportunities for its volunteers. Advanced training is available to all of its volunteers and there is strong encouragement for them to further their skills either locally or through outside programs.

EMS SYSTEM ORGANIZATION TODAY

The borough EMS program now is a unified system providing advanced level services. All administration is under the direction of the EMS Coordinator, who also is responsible for overall operational control and direction. Within each ambulance service, there also is an internal chain of command with one person designated as the service chief.

An appointed advisory board reports to the mayor and assembly on issues relating to EMS and health care within the borough. The board is made up of a representative from each ambulance service, Valley Hospital in Palmer, fire services, the Borough dispatch center, the dive rescue team, the Borough EMS Medical Director, and the Borough EMS Coordinator. The board meets monthly to discuss various EMS issues, to recommend policies and program directions, and to serve as an open forum for the exchange of new ideas and suggestions to improve the system.

Medical direction currently is provided through a contract with Frontier Emergency Physicians in Palmer. Under the direction of Dr. Janet Smalley, four physicians provide site visits to the EMS squads, assist with continuing medical education and teaching, and monitor the program for compliance with acceptable medical standards.

EMS SYSTEM EVOLUTION

The evolution of the Mat-Su Borough EMS system resulted from a strong commitment by the borough to use tax funds to support growth of the system, as well as the willingness of scores of borough citizens to volunteer their time to get training and serve on ambulance or first responder squads.

Currently, the borough is providing over $700,000 per year to support this system, which is supplemented by state funds and fees for service. Borough funds are used to pay the salaries of the EMS Coordinator and Trainer, plus funding for the volunteer services and the Palmer Dispatch Center. State funds support a clerical position and mini-grants to the volunteer ambulance services.

Today, all highway communities in the borough are served by an ambulance service operating at least at the Emergency Medical Technician (EMT) II level, and EMT-III level service is provided by seven of the ten ambulance services. The three services without EMT-IIIs are backed up by EMT-III level services.

In addition to the ambulance services, there are first responder services along remote stretches of highway, and in the two small off-highway communities of Gold Creek and Skwentna. Additional services are provided by fire departments which provide rescue services and can serve as first responders. Water rescue is provided by a specially trained dive rescue team.

Dispatching is provided from a central dispatch center in the Palmer Police Department. Dispatchers answer incoming 911 lines and dispatch appropriate resources for Mat-Su Borough EMS and fire services, as well as the Palmer Police Department. All ten dispatchers have been trained in the new Emergency Medical Dispatchers course. Additionally all dispatchers are required to have current CPR certification, and most have completed the Emergency Trauma Technician (ETT) first responder course. This training is helpful because it trains dispatchers to give emergency first aid instructions to callers while the ambulance is enroute.

With a large, widely scattered system like the Mat-Su Borough's, the ability to maintain a constant one-on-one contact between each EMT and the Medical Director is virtually impossible. The result is that it may be months before an EMT is able to meet with the Medical Director to review a particular case.

To reduce this time, the Borough EMS Coordinator has arranged to have a brief review at the hospital following all serious ambulance runs. This allows for immediate feedback to the crew and for the physicians to assess any needs they see for future training. In the coming year, the goal is to expand this policy to have the EMTs spend some time in the emergency department learning that side of the system and to get to know the Emergency Medicine physicians better.

The ability to perform routine system analysis is important both for training as well as for quality assurance. Beginning in 1988, all ambulance run reports are being entered into a central computer data base. Approximately 80 different parameters are being collected and analyzed. This will enable the EMS Coordinator and physician sponsors to look at compliance with standing orders, response times, procedures used, and locations of calls. Base line demographics information also will be collected and reviewed. This will be used to modify the training program and possibly to revise procedures and medications used on the ambulances.

The basis of the borough EMS system is its dedicated group of volunteers, ranging from drivers to EMT-IIIs, rescue technicians, firefighters, and instructors. Without this group of approximately 450 volunteers, there would be no prehospital EMS system. To maintain a high level of interest and commitment among the volunteers, the administration must work hard to provide the training and support they need. Training entails not only the initial training required for initial state certification, but also entails advanced level courses and continuing medical education necessary for recertification every two years. In addition, specialty classes are provided to help maintain interest and to teach new techniques and concepts. The size of the system means extensive commitment must be made in time and money each year to meet these needs. Of all the programs offered by the borough for the EMS system, training receives the highest priority for time and funds.

Figure 1.

Location of Ambulance and First Responder Services in the Mat-Su Borough

Ambulance Services	First Responder Services
Sutton	Skwentna
Butte	Gold Creek
Palmer	Lakes/Fishhook
Wasilla	Lake Louise/Nelchina
Houston	Eureka
Willow	Glacier View
Talkeetna	Petersville
Trapper Creek	
Mat-Su Transport Team	

FUTURE GOALS AND PRIORITIES

A priority concern for the future is the number of refresher courses needed to keep the skill levels and state certification of the volunteers up to date. As in the past, the EMS Coordinator and EMS Trainer will identify areas of greatest need and these topics will receive extra attention.

The second area of concern is the need for advanced training in pediatric emergencies. In fiscal year 1988, a pediatrics emergency course was held and, in fiscal year 1989, two pediatric advanced life support courses will be held for EMT-IIIs and nurses.

The third area of concern is for critical incident stress debriefing counseling to help the EMTs cope with major EMS incidents, such as mass casualty disasters, the death of a friend, or the death of a child. The borough already has begun such a program using trained counselors from local services and a local chaplain program. This system will be expanded in fiscal year 1990 to have additional trained people available on call to respond to the needs of the volunteers. This program also has been implemented at Valley Hospital and has been well received by families and staff.

Another priority concern is risk management. This entails looking at all aspects of the system to see where liability exposure is, and evaluating methods to reduce it. This will begin with driver training for all ambulance drivers, and a review of driver records.

SUMMARY

With a combination of local borough funds, state grant funds, and fees for service, the Mat-Su Borough has developed a modern, state-of-the-art EMS system, utilizing volunteers, which responded to over 2200 emergency calls in 1987, a 56% increase in calls over 1983. As a result, citizens and visitors in the Mat-Su Borough have a much better chance of surviving a medical emergency than they did a few short years ago.

Epidemiologic Surveillance of Enteric Diseases in Alaska -- Value of Case Investigation

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ABSTRACT

Patients infected by Salmonella, Shigella, Giardia, Campylobacter, Yersinia, and Hepatitis A virus were interviewed during a 15-month period to evaluate the benefit of intensive follow-up, to assess the proportion of cases who had contact with children less than four years of age, and to determine risk factors. Of 746 reported infections, we interviewed 345 patients (46%) and identified 199 other ill people. Follow-up of sporadic, passively reported cases required considerable resources, but no large outbreaks were uncovered. Investigation of patients [N=103 (43%)] who had contact with children less than four years of age, or children who attended daycare centers often resulted in identification of additional illness, especially for cases of giardiasis. Primary health care providers can play a key role in reducing enteric disease by increasing efforts to identify illness among daycare and family contacts of their patients.

INTRODUCTION

Enteric pathogens cause significant morbidity among Alaskans (1). Some cases are determined to be outbreak-associated, but many are sporadic in nature. Although numerous outbreaks of disease caused by enteric pathogens are well documented in the literature (2-7), studies focusing on endemic disease are fewer (8-11).

During a 15-month period, we studied enteric diseases caused by six pathogens--Salmonella, Shigella, Giardia, Yersinia, Campylobacter, and Hepatitis A virus. The purposes of the study were:

1) to evaluate the benefit of intensive follow-up of reported cases to uncover outbreaks of enteric disease and to find other cases,
2) to assess the proportion of cases of enteric disease associated with contact with children less than four years of age, either at home or through day care centers/baby-sitting contacts,
3) to determine the proportion of cases that could not be attributed to contact with small children, and
4) to determine risk factors associated with these six enteric diseases.

METHODS

Alaska law requires that health care providers report suspected or confirmed cases of infections caused by Salmonella, Shigella, Giardia, Campylobacter, Yersinia, and Hepatitis A virus to the Section of Epidemiology (12). During a 15-month period, from October 1984 through December 1985, we attempted to interview every person reported. We also attempted to interview people identified as a result of that interview.

A case was defined as an ill individual reported in accordance with Alaska law who we interviewed, plus individuals named during our interview with the index case who we interviewed and were also ill.

A standard data collection form was used to conduct all interviews. In addition to demographic information, we collected data on symptoms, known or possible risk factors such as related household illness, contact with children less than four years of age (at home, through a day care center or a baby sitting encounter), food history, water consumption history, history of travel outside Alaska, contact with pets or farm animals, and antacid use. Individuals with hepatitis A were also questioned about occupations and sexual orientation.

If the case was associated with a day care center (DCC) or baby sitting service, either directly or indirectly as a household member, we contacted the DCC or baby sitter to determine if similar illness existed there. We requested stool samples from possible cases, household members, DCC and baby sitting attendees.

Stool samples were submitted to the state laboratory using Carey-Blair transport medium for bacterial culture and 10% formalin for ova and parasites. In investigations of hepatitis A, we requested a blood sample on possible cases and all household contacts of confirmed hepatitis A cases. Blood specimens were analyzed for hepatitis A IgM antibody at the State Public Health Laboratory-Fairbanks, Section of Laboratories, Division of Public Health.

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All cases were entered into our computerized infectious disease database. Cases and possible cases who were epidemiologically related were each assigned unique investigation numbers.

To assess the representativeness of cases, we compared their age, sex, race, and geographic location to the 401 passively reported, not interviewed people (Table 1). Persons living in Anchorage, Juneau, Fairbanks, or Ketchikan were classified as urban; all others were classified as rural. In order to compare by race, we assigned race to the 401 not-interviewed people whose race was unknown according to place of residence. Urban areas were assigned non-Native status, while all other cases were assigned Native status.

Table 1. Case definitions and categories

CASES		345	345	345
Named with symptoms, not interviewed		199		
Passively reported by health care providers	(27)*			
Not otherwise identified	172			172
Number found through case investigations		544		
Passively reported, not interviewed			401	401
Named during interviews	(27)*			
Number reported in state statistics			746	
Total possible cases				918

*Same people

RESULTS

We identified 746 cases of enteric illness that were included in official statistics, and we interviewed 345 (46%); we did not interview 401 (Table 1).

As a result of these 345 interviews, 199 additional individuals were named and reported to have had similar illness. Because illness among 172 (86%) of the 199 could not be independently confirmed, they were not counted in official statistics. Twenty-seven were found to have been reported independently by a primary health care provider and were counted in official statistics, although not interviewed by us (Table 1).

For purposes of our study, we defined cases to include only the 345 ill and interviewed people. The 345 interviewed cases and 401 not-interviewed people reported in state statistics were similar in age (median age 28 years vs. 25 years), sex (males 47% vs. 49%), race (Native 5% vs. 10%), and geographic location (urban 76% vs. 75%).

The 544 ill individuals represented 188 investigations (Table 2). Of the 188 investigations, 85 (45%) involved Giardia, 38 (20%) Salmonella, and 37 (20%) Campylobacter. Shigella, Hepatitis A, and Yersinia each made up less than 10% of investigations (Table 2 and Figure 1). Of the 19 Hepatitis A investigations, three involved clusters of 5, 7 and 8 cases, respectively.

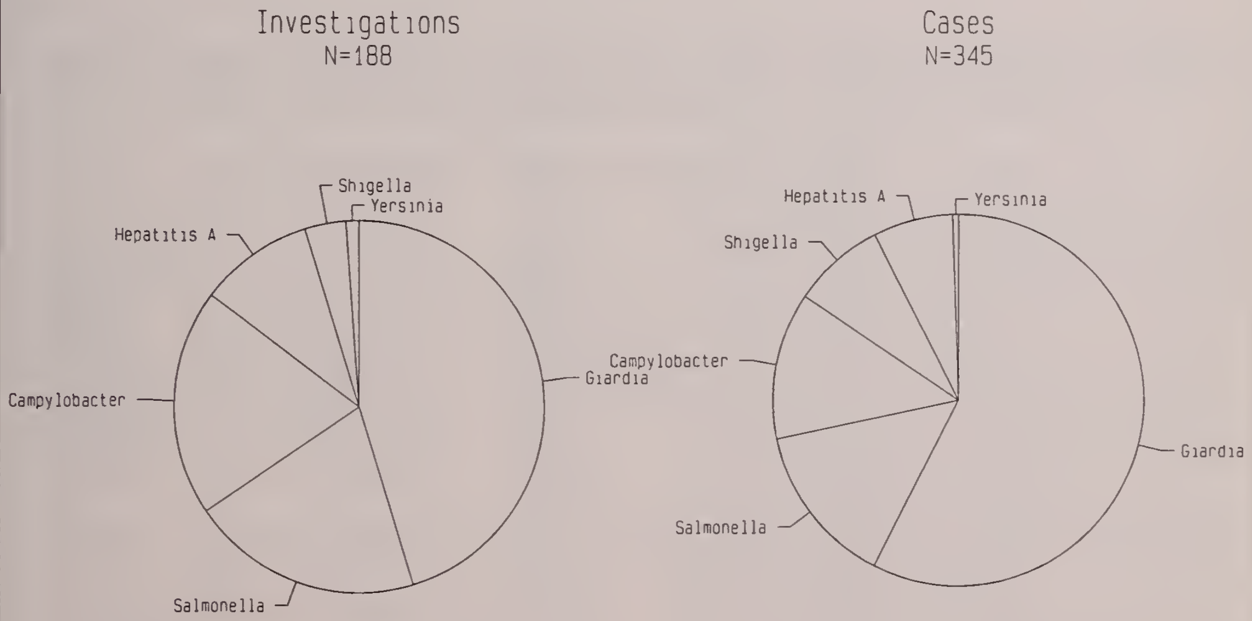
For the 188 investigations, the number of ill individuals identified per investigation ranged from 1 to 141. Of the 188 investigations, 100 (53%) identified 1 ill individual, 42 (22%) identified 2 ill individuals, 39 (20%) identified 3-6 ill individuals. Two investigations identified 7 ill individuals (Campylobacter, Hepatitis A), 2 identified 8 ill individuals (Giardia, Hepatitis A), and one cluster of 9 people with Giardia was found. Two investigations involved large common source outbreaks--a foodborne shigellosis outbreak affecting 25 people and a waterborne giardiasis outbreak affecting 141 people.

Of the 345 cases, 280 (81%) were laboratory-con-

Table 2. Individuals ill per investigation; possible cases per case

	Number of Cases	Named, Ill Not Interviewed	Total	Number of Investigations	Number Ill Per Case
Salmonella	49	23	72	38	.47
Shigella	28	9	37	7	.32
Giardia	198	127	325	85	.64
Campylobacter	44	22	66	37	.50
Hepatitis A	24	18	42	19	.75
Yersinia	2	0	2	2	-
Total	**345**	**199**	**544**	**188**	**.58**

Figure 1. Investigations and Cases by Disease



firmed, ranging from 64% of cases of shigellosis to 100% of cases of yersiniosis. Of the 345 interviewed cases, 198 (58%) had illness caused by Giardia, 49 (14%) by Salmonella, and 44 (13%) by Campylobacter. Shigella and Hepatitis A virus each caused less than 10% of illnesses. Yersinia caused only two cases (Figure 1 and Table 2). Of the 401 cases not interviewed, 282 (70%) had illness caused by Giardia, 54 (13%) by Salmonella, and 48 (12%) by Campylobacter. Shigella, Hepatitis A virus, and Yersinia each caused less than 10% of cases.

Of the 345 cases, 71 (20.5%) occurred in the 0-4 year age group (Table 3). Of the 49 cases of salmonellosis, 13 (26%) occurred in the 0-4 age group. Although 29% of shigellosis cases occurred in the 0-4 age group, one common-source outbreak of shigellosis accounted for 25 of the 28 cases. Of the 24 cases of Hepatitis A, 20 (83%) occurred in people older than 19 years; and 75% of Campylobacter cases were also seen in adults older than 19 years. Two age groups (0-4 and 30-39) accounted for 23% and 30%, respectively, of the cases of giardiasis, for both sporadic cases and cases associated with a large common-source, waterborne outbreak (Table 3 and Figure 2). Median ages were 21 years for salmonellosis, 26 years for shigellosis, 30 years for hepatitis A and giardiasis, and 30.5 years for campylobacter infection.

Of the 345 cases, 162 (47%) were male (Table 4). Of the 345 study cases, 310 (90%) occurred in non-Natives, 28 (8%) occurred in Native Alaskans, and 7 (2%) were of unknown race. Of the 310 non-Native cases, 296 (95%) were Caucasian, 6 (2%) were Black, and 4 (1.3%) each were Asian or Hispanic. Non-Natives accounted for 80% of salmonella cases, 95% of campylobacter cases, and 92% of hepatitis A cases (Table 4). Of the 345 cases, 260 (76%) resided in urban areas (Table 4).

Two pathogens, Campylobacter and Giardia, showed marked summer peaks in 1985. Cases of Campylobacter infection showed the most dramatic summer peak, whereas cases of giardiasis remained elevated through October. Cases of salmonellosis had a slight summer peak which appeared to be part of a cyclic trend throughout the year (Figure 3). The large peak of giardiasis in fall 1984 to January 1985 was due to a large, common-source, waterborne outbreak. The December 1984 shigellosis peak was due to a common-source, foodborne outbreak.

Diarrhea was a prominent symptom for Salmonella, Shigella, Giardia, and Campylobacter infections, but less than half of the cases of hepatitis A reported diarrhea. Half of the cases of salmonellosis reported bloody diarrhea. Fifty-six percent of cases of giardiasis reported bloating, and 78% reported gas. Fever was

Table 3. Number and Percent (%) of cases by age group

Age Group	Salmonella N (%)	Shigella N (%)	Giardia N (%)	Campylobacter N (%)	Hepatitis A N (%)	Yersinia N (%)	Total N (%)
0-4	13 (27)	8 (29)	46 (23)	4 (9)	0 (0)	0 (0)	71 (21)
5-9	3 (6)	4 (14)	11 (5.5)	3 (7)	1 (4)	0 (0)	22 (6)
10-14	2 (4)	1 (3.5)	10 (5)	2 (4.5)	1 (4)	1 (50)	17 (5)
15-19	5 (10)	0 (0)	6 (3)	2 (4.5)	2 (8)	0 (0)	15 (14)
20-24	5 (10)	1 (3.5)	12 (6)	0 (0)	1 (4)	0 (0)	19 (5.5)
25-29	7 (14)	5 (18)	11 (5.5)	10 (23)	6 (25)	0 (0)	39 (11)
30-34	2 (4)	5 (18)	30 (15)	7 (16)	3 (12.5)	0 (0)	47 (14)
35-39	3 (6)	1 (3.5)	29 (15)	3 (7)	0 (0)	0 (0)	36 (10)
40-44	1 (2)	0 (0)	10 (5)	2 (4.5)	0 (0)	0 (0)	13 (4)
45-49	3 (6)	1 (3.5)	12 (6)	1 (2)	6 (25)	0 (0)	23 (7)
50-54	0 (0)	1 (3.5)	9 (4.5)	2 (4.5)	3 (12.5)	0 (0)	15 (4)
55-59	0 (0)	1 (3.5)	7 (3.5)	5 (11)	0 (0)	0 (0)	13 (4)
60-64	2 (4)	0 (0)	4 (2)	1 (2)	0 (0)	1 (50)	8 (2)
65-69	1 (2)	0 (0)	0 (0)	1 (2)	0 (0)	0 (0)	2 (0.5)
70+	2 (4)	0 (0)	1 (0.5)	1 (2)	1 (4)	0 (0)	5 (1.5)
Total	**49**	**28**	**198**	**44**	**24**	**2**	**345**

Figure 2. Age of Cases by Disease
N=343 (2 cases Yersinia not shown)

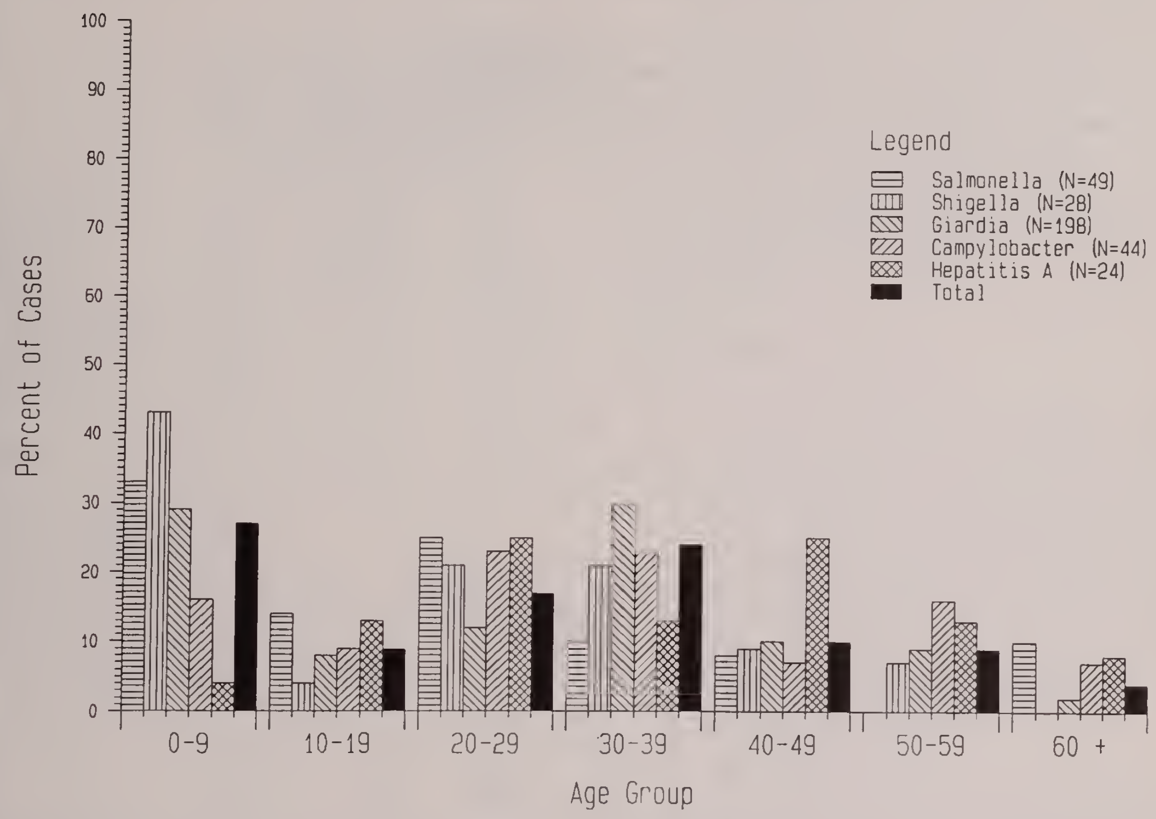


Table 4. Number and percent (%) of cases by sex, race and geographic location

	Male N (%)	Native N (%)	Urban* N (%)	Total N
Salmonella	23 (47)	10 (20)	25 (51)	49
Shigella	14 (50)	1 (4)	26 (93)	28
Giardia	92 (46)	14 (7)	161 (81)	198
Campylobacter	17 (39)	1 (2)	34 (77)	44
Hepatitis A	14 (58)	2 (8)	14 (58)	24
Yersinia	2 (100)	0 (0)	2 (100)	2
Total	**162 (47)**	**28 (8)**	**260 (76)**	**345**

*Urban = Anchorage, Fairbanks, Juneau, and Ketchikan

reported by 76% of cases of salmonellosis and 75% of cases of campylobacter enteritis. Vomiting was reported by 63% of cases of hepatitis A (Table 5). Duration of symptoms averaged eight days for cases of shigellosis and campylobacter infection, 14 days for cases of salmonellosis, 21 days for cases of hepatitis A, and 24 days for cases of giardiasis.

Individuals infected by campylobacter were more likely to have had contact with a farm animal. Travel outside Alaska prior to illness was reported by 67% of patients with hepatitis A, including three with a history of travel to Mexico (Table 6).

Individuals with giardiasis were most likely to have reported consumption of surface water (Table 7) and DCC attendance for a patient or family member (Table 8).

Illness similar to that of the index case was found in 15 (48%) of DCCs named by a case, including 11 (48%) DCCs connected to cases of giardiasis (Table 9).

As a result of our investigation of the reported index case, we recommended obtaining a stool sample from 370 ill individuals; 199 were submitted and 85 (43%) were positive (Table 10). Of the 117 submitted for giardiasis, 24 (29%) of 82 not associated with a single large, common-source outbreak were positive.

Figure 3. Cases by Month and Year of Onset by Disease
N=343 (2 cases of Yersinia not shown)

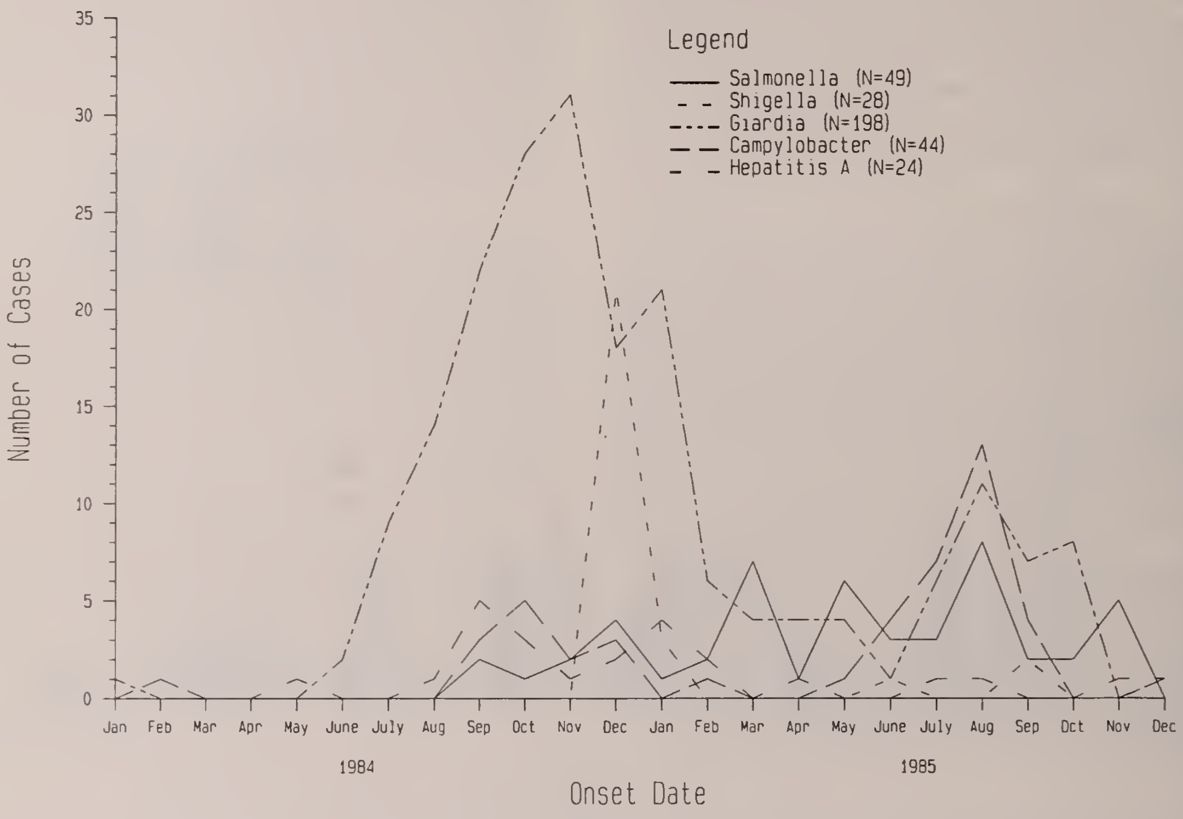


Table 5. Percent of cases reporting each symptom* (N - 343)

Symptoms	Salmonella (N = 49)	Shigella (N = 28)	Giardia (N = 198)	Campylobacter (N = 44)	Hepatitis A (N = 24)
Abdominal cramps	71	71	81	89	42
Bloating	27	14	56	34	--
Chills	59	36	28	73	63
Diarrhea	100	96	90	98	42
-bloody	51	17	7	32	--
-mucus	33	36	22	34	4
-watery	90	86	75	84	25
Excessive gas	41	40	78	50	--
Fatigue	76	43	71	80	88
Fever	78	43	28	75	63
Headache	45	36	39	55	50
Joint pains	31	18	18	45	42
Loss of appetite	80	57	69	75	--
Muscle aches	49	21	35	61	71
Nausea	61	21	55	57	58
Vomiting	43	21	34	32	63
Weight loss	78	36	55	69	67

* Two cases of yersinia not shown

Table 6. Risk factors

	Travel* N (%)	Owned a pet N (%)	Owned a farm animal N (%)	Total N
Salmonella	10 (20)	23 (47)	2 (4)	49
Shigella	5 (18)	14 (50)	1 (4)	28
Giardia	44 (22)	136 (69)	19 (10)	198
Campylobacter	6 (14)	29 (66)	14 (32)	44
Hepatitis A	16 (67)	- -	- -	24
Yersinia	0 (0)	1 (50)	1 (50)	2
Total	**81 (23)**	**203 (64)**	**37 (12)**	**345**

*Travel outside Alaska

Table 7. Type of water* consumed within 2 months of illness

	Municipal N (%)	Well N (%)	Surface** N (%)	Total N
Salmonella	37 (76)	17 (35)	9 (18)	49
Shigella	5 (18)	13 (46)	0 (0)	28
Giardia	169 (85)	53 (27)	65 (33)	198
Campylobacter	29 (66)	26 (59)	10 (23)	44
Hepatitis A	21 (88)	5 (21)	2 (8)	24
Yersinia	2 (100)	0 (0)	0 (0)	2
Total	**263 (76)**	**114 (33)**	**86 (25)**	**345**

*Cases may have consumed more than one type of water or may not have consumed any of the above types of water.

**Surface = river, spring, or stream.

CONTACT WITH CHILDREN LESS THAN 4 YEARS OF AGE

Of the 345 cases, 71 (21%) were among infants in the 0-4 year age group. Of 238 patients older than 12 years of age, 75 (32%) reported contact with an ill child in the 0-4 year age group. If the two large common-source outbreaks (Giardia and Shigella) are eliminated from the analysis, the proportion of remaining patients reporting contact with an ill child was similar.

Of the 238 cases older than 12 years of age, 103 (43%) reported contact with children less than 4 years of age, whether ill or not (Table 11). When all cases are analyzed together, patients age 12 or less (89/107, 83%) were more likely to have contact with a child less than 4 years of age than patients older than 12 years (103/238, 43%) (Chi square $p < .0001$). When analyzed by disease, this difference remained only for giardiasis (Chi square $p < .0001$) and salmonellosis (Chi square $p < .0006$).

DISCUSSION

Giardia was the most frequently reported enteric pathogen (even without the large waterborne outbreak) followed by Salmonella and Campylobacter. Shigella, in the absence of a single common-source outbreak, was infrequently reported. Cases of hepatitis A also were few during the study period, in marked contrast to the current statewide epidemic (13). Only two cases of yersiniosis were interviewed and five were reported during the 15-month study period.

Campylobacter has emerged as an important enteric pathogen. Patterns of infection in Alaska are similar to national patterns with a marked increase in the summer and a strong association with contact with farm animals (14,15).

Symptoms were similar to those reported in other studies. Diarrhea was prominent in all disease except hepatitis A. Vomiting was more prominent in individuals with hepatitis A. Bloody diarrhea was reported frequently by patients with salmonellosis (51%) and campylobacter enteritis (32%). Fever frequently was present among cases of salmonellosis (78%) and campylobacter enteritis (75%). Bloating and excessive gas were reported more frequently by individuals with giardiasis. Physicians and health-care providers evaluating patients with symptoms of bloating and excessive gas, especially if present for more than seven days, should obtain a stool sample for ova and parasites to detect Giardia.

Enteric diseases cause significant morbidity and loss of productive work. Duration of symptoms averaged eight days for cases of shigellosis and campylobacter enteritis, 14 days for salmonellosis, 21 days for hepatitis A, and 24 days for giardiasis.

Overall, 25% of the cases reported drinking un-

Table 8. Number of cases with family members attending day care*

	Yes N (%)	No N (%)	Unknown N (%)	Total N
Salmonella	4 (8)	45 (92)	0 (0)	49
Shigella	6 (21)	22 (79)	0 (0)	28
Giardia	55 (28)	138 (68)	5 (3)	198
Campylobacter	1 (2)	43 (98)	0 (0)	44
Hepatitis A	0 (0)	24 (100)	0 (0)	24
Yersinia	0 (0)	2 (100)	0 (0)	2
Total	**66 (19)**	**274 (79)**	**5 (1)**	**345**

*Includes cases who attended day care

Table 9. Follow-up of daycares for similar illness

	Illness Found N (%)	Total Number of Daycares N
Salmonella	2 (50)	4
Shigella	1 (33)	3
Giardia	11 (48)	23
Campylobacter	1 (100)	1
Hepatitis A	0	-
Total	**15 (48)**	**31**

treated surface water. Among those with giardiasis, 33% of cases drank untreated surface water. Recently, another study showed an increased risk of giardiasis among individuals who drank surface water or water from a shallow well (8). The 141 individuals who became ill with giardiasis in a large waterborne outbreak were also, in a sense, victims of "untreated surface water" because the outbreak occurred when the chlorinator malfunctioned at one of the tank houses which drew water from a surface lake (16).

Most cases of enteric disease could not be linked to a day care center. However, on investigation of those with DCC contacts, we found reports of 15 DCCs with similar illness. In 11 (78%) of these DCCs, illness was due to Giardia. Of the 198 cases of giardiasis, 55 (28%) reported DCC attendance. This finding was not unexpected. Our earlier study found undetected cases of giardiasis in two DCCs (17), and DCC attendance is a well-known risk factor for giardiasis (18). Because of this strong association, aggressive follow-up of cases of giardiasis identified in DCCs is essential. We recommend that all clinicians ask patients about contact with children less than four years of age. Additional investigation should concentrate on children, their families, and any associated DCC. All family members and ill contacts should submit a stool sample for identification of Giardia.

When we requested stool samples from contacts, compliance was high (54%) and tests were productive (43% positive). Follow-up with family members and ill contacts identified other cases, enabling them to be appropriately diagnosed, counselled, and treated.

We were able to find more ill contacts when a child less than four years of age was involved. Asking a person with a case of enteric disease whether they have had contact with a child less than four years of age was a useful screening tool for evaluating the need for further follow-up.

Although general risk factors associated with these six enteric diseases are well known, specific sources were not identifiable. Even if all transmission from young children (child to child, child to adult) were eliminated, a substantial number (and proportion) of enteric disease cases would remain. More than one-half of all patients older than 12 years of age reported no contact with children, including nearly two-thirds of cases of salmonellosis (68%) and hepatitis A (64%).

The fact that we were able to identify an ill contact for every two cases strengthens the notion that many persons with enteric diseases are never diagnosed and thus never reported. Persons may more likely obtain medical care if their illness is severe or if they are part of a recognized outbreak.

Nearly three-fourths of our investigations yielded only the index case or the index case plus one other individual. Follow-up of sporadic cases required considerable effort and no large outbreaks were uncovered. Only two large outbreaks were identified during the study, both were initially reported as possible outbreaks, not as sporadic cases of individual illness.

Our study suggests that when follow-up of a case of enteric disease shows there has been contact with children under 4 years of age in the household or a link to a DCC or other household illness, cases should be investigated intensively. Targeting intervention in these circumstances will increase the likelihood of identifying other cases in need of appropriate evaluation,

Table 10. Follow-up of possible cases with a request for stool samples

	Number Positive N	Percent Positive (%)	Number Submitted N	Number Recommended N
Salmonella	10	20*	49	85*
Shigella	21	70	30	41
Giardia	54	46	117	233
Campylobacter	0	0	3	11
Yersinia	0	0	0	0
Total	**85**	**43**	**199**	**370**

*Included 25 (all negative) requested during investigation of two cases in a village.

Table 11. Contact with any child less than four years of age by cases greater than 12 years of age (N=238)

	Yes N (%)	No N (%)	Total
Salmonella	10 (32)	21 (68)	31
Shigella	12 (80)	3 (20)	54
Campylobacter	15 (42)	21 (58)	36
Hepatitis A	8 (36)	14 (64)	22
Giardia	58 (44)	75 (56)	67
Yersinia	0 (0)	1 (100)	1
Total	**103 (43)**	**135 (57)**	**238**

diagnostic tests, treatment, and education. Such targeted intervention will reduce the burden of enteric disease in Alaska.

SUMMARY

1. Investigations of cases who had contact with children less than four years of age or with children who attended daycare centers often resulted in identification of additional enteric illness, especially giardiasis.

· Ill contacts were identified more often for cases of giardiasis. All cases should be investigated and those with illness should be tested by stool examination or empirically treated.

· 33% of patients with giardiasis drank untreated surface water. All surface water should be boiled prior to drinking.

2. Many cases were found to be sporadic; devoting time or resources to follow-up of sporadic, no child-associated cases will uncover few new cases.

3. In spite of investing considerable resources of time and personnel, many investigations were unsuccessful because the index case could not be contacted. Primary health care providers could be especially effective in implementing our recommendations at the time they initially evaluate and treat patients.

· 56% of individuals on whom we requested stools returned a stool sample for analysis. Physicians should routinely test patients' stools for bacterial and parasite infections.

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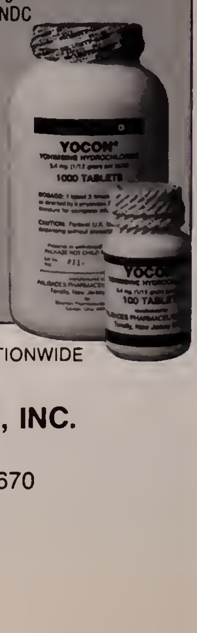

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Special Feature

Dr. Oleg Balanov is the Deputy Director of the Bekhterev Psychoneurological Research Institute which was founded in 1907 by Vladimir Bekhterev, a prominent Russian neurologist, psychiatrist, and psychologist. The Institute, basically, deals with clinical psychological studies and mental and neurological disorders as well as the rehabilitation of patients with both neurological and mental disorders. Dr. Balanov heads the Department dealing with post-stroke rehabilitation both on an in- and out-patient basis. He was in Alaska in 1985 and will be returning to the state in September of 1990 as the guest of the Institute for Circumpolar Health Studies of the University of Alaska Anchorage.

The following manuscript is exactly as received from Dr. Balanov; first in the Cyrillic alphabet, followed by the English translation.

СССР

ЛЕЧЕНИЕ ДЕПРЕССИЙ У БОЛЬНЫХ ПОСЛЕ ИНСУЛЬТА

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В теоретическом отношении понятие непсихотическое депрессивное состояние у постинсультных больных - "постинсультная депрессия" - является малоизученной разновидностью органической депрессии. В то же время депрессивные состояния, по мнению ряда авторов (8,9) отмечаются спустя 2-6 месяцев после инсульта у 40-60% больных.

В этой работе мы не касаемся важной роли психосоциальных и психокоррекционных методов лечения непсихотических депрессий у больных после инсульта, без чего теперь не обходится система их комплексного лечения (I-5,9,II), а ограничили свою задачу исследованием их выявления и действия медикаментозных средств.

Нами было исследовано 28% (90) больных, перенесших инсульт, с непсихотическими депрессивными состояниями (мужчин - 57, женщин - 33). Возраст их варьировал от 32 до 84 лет (в среднем 55,9 года). Больные были разделены на 3 группы, по 30 человек в каждой. В первую группу включены 30 больных, получавших антидепрессанты типа амитриптилина, во вторую группу - 30 больных, получавших малые транквилизаторы (седуксен или сибазон) и в третью (контрольную) - 30 больных, не получавших ни антидепрессантов, ни транквилизаторов. Характер неврологических нарушений верифицировался квалифицированными невропатологами. У 64 больных патологический очаг локализовался в одном из полушарий мозга (3I слева, 33 справа), у 26 - в стволе мозга. У 79,7% (5I) больных с локализацией очага в полушарии мозга имелись гемиплегии или гемипарез. У 6I,5% (I6) больных с локализацией очага в стволе отмечен гемипарез. Таким образом, у большинства больных установлены выраженные двигательные нарушения.

Депрессивные состояния характеризовались маловыраженным или умеренным подавленным настроением в форме немотивированной или маломотивированной тревоги, иногда с компонентом маловыраженной тоски. У 82,2% (74) больных настроение значительно улучшалось к вечеру, у остальных 7,8% (16) - оставалось прежним. У больных не отмечено чувства вины, суицидных попыток, выраженной заторможенности мышления, беспомощности, безнадежности и никчемности. Наследственность у больных не отягощена психическими заболеваниями. У больных до возникновения инсульта не имелось каких-либо заметных и продолжительных (более 1-2 дней) состояний подавленного или приподнятого настроения. Наряду с клинико-психопатологическим методом исследования больных, использовалась и шкала депрессии Гамильтона (7); результаты подвергнуты статистической обработке по методу Стьюдента-Фишера.

Шкала депрессии Гамильтона с успехом используется не только при психотических, но и непсихотических, в том числе "постинсультных депрессиях". Этой шкалой могут пользоваться не только психиатры, но и невропатологи.

Амитриптилин назначался внутрь по схеме: 1-я неделя по 0,025 в сутки, 2-я - 3-я недели по 0,05 и 4-я неделя - по 0,075 в сутки. Седуксен назначался: 1-я неделя по 0,005 в сутки, 2-я - 3-я недели по 0,01 в сутки, 4-я неделя - по 0,015 в сутки.

Результаты исследования

Начальный уровень выраженности депрессивных состояний у больных, получавших амитриптилин (I группа) и не получавших его (3 группа) был одинаков (табл. I). Однако 4 недели спустя в первой группе депрессивные состояния не отмечались, а в третьей - сохранялись, различия между этими группами становились отчетливыми ($p < 0,001$).

Таблица I

Сопоставление выраженности депрессивных состояний у больных после инсульта, получавших амитриптилин (I группа) и не получавших его (3 группа)

Группа больных	Средняя выраженность депрессивных состояний в баллах по шкале Гамильтона ($\bar{x} \pm S_{\bar{x}}$) через интервал:	
	исходный уровень	4 неделя
Первая	I2,85±0,526	4,5±0,705
Третья (контрольная)	I3,45±0,448	9,05±0,44
Достоверность различия (p)	>0,I	<0,00I

Исходный уровень выраженности депрессий у больных I и 2 групп был идентичен (табл.2).

Таблица 2

Сопоставление выраженности депрессивных состояний у больных после инсульта, получавших амитриптилин (I группа) и седуксен (2 группа)

Группы больных	Средняя выраженность депрессивных состояний в баллах по шкале Гамильтона ($\bar{x} \pm S_{\bar{x}}$) через интервал:	
	исходный уровень	4-я неделя
Первая	I2,85±0,526	4,5±0,705
Вторая	I3,65±0,599	8,85±0,354
Достоверность различия (p)	>0,I	< 0,00I

Вместе с тем, 4 недели спустя установлены достоверные различия между больными I и 2 групп (p<0,001).

Таблица 3

Сопоставление выраженности депрессивных состояний у больных после инсульта, получавших седуксен (2 группа) и контрольной (3-й группы)

Группы больных	Средняя выраженность депрессивных состояний в баллах по шкале Гамильтона ($\bar{x} \pm S_{\bar{x}}$) через интервал:	
	исходный уровень	4-я неделя
Вторая	I3,65±0,599	8,85±0,354
Третья (контрольная)	I3,45±0,448	9,05±0,44
Достоверность различия (p)	>0,I	>0,I

Как следует из табл.3, не получено достоверных различий ($p>0,I$) между уровнем выраженности депрессивных состояний при назначении седуксена (2-я группа) и без применения транквилизаторов и антидепрессантов (3-я группа). Курсовое применение седуксена не способствовало заметному снижению депрессивного состояния. У 28,4% больных, получавших седуксен, появились наряду с чувством мышечной слабости ипохондрические фиксации на своих ощущениях в голове, теле или конечностях, хотя ипохондричности в преморбиде не отмечалось. Наши данные об эффективности применения антидепрессантов типа амитриптилина при непсихотических депрессивных состояниях после церебрального инсульта (I0) совпадают с результатами, полученными другими исследователями, применявшими амитриптилин или нортриптилин (8,9).

Роли астено-депрессивных состояний у больных, перенесших инсульт, понижающих психическую и двигательную активность, уделили внимание ряд авторов (3). В отличие от общепринятого применения малых транквилизаторов при лечении непсихотических депрессивных состояний, по нашим данным, начиная со 2-го месяца, показано назначение антидепрессантов типа амитриптилина. Нельзя согласиться с положением о специфических расстройствах настроения при инсульте (6). По нашим данным особой специфичности расстройств настроения при инсульте не наблюдается.

Ввиду недостаточности количества исследованных больных (I5,5%), проживающих в Заполярье или Сибири, не представилось возможным проведение у них математическо-статистической обработки полученных результатов. Однако эти исследования вскоре будут завершены.

На основании полученных данных можно сделать следующие выводы:

I. Непсихотические депрессивные состояния у больных после инсульта могут быть выявлены с помощью шкалы депрессии Гамильтона.

2. Для лечения непсихотических депрессивных состояний у этих больных целесообразно применение антидепрессантов типа амитриптилина по изложенной схеме.

3. Курсовое применение малых транквилизаторов (седуксен и др.) не только не вызывает заметного ослабления выраженности депрессивных состояний, но из-за миорелаксации у 28,4% больных вызывает или усугубляет ипохондрические фиксации.

О. Садов

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О. Садов

Therapy of Depressions in Post-Stroke Patients

by O.A. Balunov(1)
O.G. Sadov
A. Yu. Alemasova

SUMMARY

In a study of 90 patients with post-stroke depression the Hamilton Depression Scale was used together with a clinical psychopathological study. One group of patients was treated with amitriptyline, another with minor tranquilizers (seduxen), and the third (controls) received neither antidepressants, nor tranquilizers. Only treatment in the first group was effective, i.e. depressions were reduced after therapy with amitriptyline So amitriptyline was demonstrated to be the most effective drug of choice in the treatment of non-psychotic post-stroke depressions.

INTRODUCTION

Theoretically, the notion of a non-psychotic depressive state, “a post-stroke depression’, is a variant of organic depression that has not yet been sufficiently studied. At the same time, such depressive states have been noted in 40 to 60 per cent of all patients some 2-6 months after stroke (8,9).

In this article the authors do not deal with a significant role of psychosocial techniques and therapeutic modification which are currently inevitable within the compound system of treatment of non-psychotic depressions in post-stroke patients (1-5,9,11); their goal was limited to a consideration of the depression identification and the effects of medication on those depressions.

The authors studied 90 (57 males and 33 females), or 28 percent of the post-stroke patients, who developed non-psychotic depressive states. The patients’ age was 32 to 84 years (average age of 55.9 years).

The group was subdivided into three subgroups of 30 patients each. Subgroup 1 comprised patients who received antidepressants of amitriptyline type, in Subgroup 2 patients received minor tranquilizers (seduxene or sibazone), and in Subgroup 3 (controls) patients received no antidepressants or minor tranquilizers.

The nature of neurological impairment was verified by qualified neurologists. In 64 subjects the pathological focus was localized in one of the brain hemispheres (31 on the left and 33 on the right side), in 26 subjects it was found to be in the brain trunk. In 51 (79.7%) patients with the lesion in the brain hemispheres, hemiplegias or hemipareses were found. In 16 (61.5%) patients with the lesion in the trunk, hemiparesis was noted. Thus, the majority of the patients revealed severe motor disturbances.

Depressive states in these patients were characterized by a weak and moderate depression of mood in the form of anxiety with no or little motivation, sometimes comprising a minor melancholy component. In 74 (82.2%) of the patients the mood significantly increased by the evening while in other 16 (7.8%) it did not change. The patients revealed no inferiority sense, suicidal attempts, severe slowing down in thinking, helplessness, hopelessness or uselessness. No patients had any hereditary risk for mental disorders. Prior to their stroke they did not manifest any notable or prolonged (over 1-2 days) states of increased or decreased mood. Together with the clinico-psychopathological method the authors used the Hamilton Depression Scale in their study of patients (7); the obtained results were statistically processed according to the Student-Fisher technique.

The Hamilton Depression Scale has been successfully applied to not only psychotic but also to non-psychotic depressions including post-stroke depressions. This scale may be useful not for psychiatrists alone, but for neurologists too.

Amitriptyline was given to patients orally according to the following scheme: 0.025 mg daily during the first week, 0.05 mg daily during the second and third weeks, and 0.075 mg daily during the fourth week. Seduxen was given in the following fashion - 0.005 mg daily (first week), 0.01 mg daily (second and third weeks), 0.015 mg (fourth week).

RESULTS

The initial level in severity of the depressive states in the patients who received amitriptyline (Group 1) was the same as in controls (Group3) (See Table 1). However, four weeks later Group 1 manifested no depressive states while Group 3 retained depression, and the difference between these two groups became evident ($p<0.001$).

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Table 1.

Comparison of severity of the depressive states in post-stroke patients who received amitriptyline (Group 1) and controls (Group 3)

Patient Groups	Average severity of depressive states in points of the Hamilton Scale $(\bar{x} \pm S\bar{x})$ with a 4-week interval	
	Initial level	final level
Group 1	12.85 ± 0.526	4.5 ± 0.705
Group 3 (controls)	13.45 ± 0.448	9.05 ± 0.44
Statistical significance of difference (p)	> 0.1	< 0.001

The initial level of the depression severity in patients of Groups 1 and 2 was identical (See Table 2).

Table 2

Comparison of severity of depressive states in post-stroke patients who received amitriptyline (Group 1) and seduxen (Group 2)

Patient groups	Average severity of depressive states in points of the Hamilton Scale $(\bar{x} \pm S\bar{x})$ with a 4-week interval	
	initial level	final level
Group 1	12.85 ± 0.599	4.5 ± 0.705
Group 2	13.65 ± 0.526	8.85 ± 0.354
Statistical significance of difference (p)	> 0.1	< 0.001

Besides, after a 4-week interval significant differences between the patients of Groups 1 and 2 was established ($p<0.001$).

Table 3.

Comparison of severity of depressive states in post-stroke patients who received seduxen (Group 2) and controls (Group 3)

Patient	Average severity of depressive states in points of the Hamilton Scale $(\bar{x} \pm S\bar{x})$ with a 4-week interval	
	initial level	final level
Group 2	13.65 ± 0.599	8.85 ± 0.354
Group 3 (controls)	13.45 ± 0.448	9.05 ± 0.44
Statistical significance of difference (p)	> 0.1	> 0.1

As Table 3 shows, no significant differences (p 0.1) was found between the severity levels in the depressive states in cases of seduxen treatment (Group 2) and the controls (Group 3), who did not receive amitriptyline or seduxen. The course-like taking of seduxen did not significantly facilitate a decrease of depression. In 28.4% of the patients who received seduxen there was found, alongside with the sense of muscular weakness, hypochondriacal fixation on one's feelings in the head, body and extremities, while no hypochondricity was found in their premorbid condition. the obtained findings of the efficacy of the amitriptyline type of antidepressants in non-psychotic post-stroke depressions of our own (10) coincide with the findings of other authors who applied amitriptyline or nortriptyline (8,9).

Some authors paid attention to the role of asthenodepressive conditions in post-stroke patients that decreased mental and motor activity (3). As distinct from the common use of minor tranquilizers in therapy of non-psychotic depressive states we have found that, starting from the second month, prescription of the amitriptyline type antidepressants was of use.

We cannot agree with the existence of specific mood disturbances in stroke.

Since the number of post-stroke patients living in Polar Regions and Siberia was not sufficient (15.5%), the authors could not provide statistical processing of the obtained findings with them. However, these studies will be finished soon.

On the basis of the obtained findings the follow conclusions can be made:

(1) Non-psychotic depressive states in post-stroke patients can be identified by means of the Hamilton Depression Scale.

(2) Effective treatment of non-psychotic depressive states in such patients needs application of the amitriptyline type antidepressants in accord with the suggested scheme.

(3) A course-like application of minor tranquilizers (seduxen etc.) does not result in any significant decrease in severity of these depressive states, but vice versa causes and increases, due to myorelaxation, hypochondriacal fixations in 28.4% of the patients.

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45th ANNUAL CONVENTION
JUNE 7 - 10, 1989

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Alaska State Medical Association
Jeanette McCart, Executive Assistant
4107 Laurel Street
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Registration Fee: $250.00 for Non-member physicians. This fee entitles physicians to attend all CME sessions. No fee is required for ASMA members.

Please register prior to May 1, 1990.

Location & Date of Meeting

Alaskaland
Fairbanks, Alaska
June 7-10, 1990

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15 hours of scientific medical education will be provided.

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Over 300 rooms have been reserved at special rates in Fairbanks.

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Excitement will be high in Fairbanks! Attendees can enjoy the University of Alaska-Fairbanks Museum, relax while cruising on the Riverboat Discovery, meander through Alaskaland Park; and for the adventureous, run for fun with Alaska's fastest (and not so fast). The grand finale to three fun-packed days will be the President's Dinner Ball.

Family Fun

A full slate of spouse and children's activities is being planned by the ASMA Auxiliary. Babysitting will be available.

Transportation

Alaska Airlines provides daily jet service from Seattle, Anchorage, and other Alaskan cities. Phone 800-426-0333 for reservations. For local info, check phone book.

Deposit/Cancellation

Non-member physicians must send $50 preregistration fee by May 1. Full refunds will be made upon request prior to May 15, 1990. No refund thereafter.

Tentative Schedule of Events
Convention '90

Thursday - June 7, 1990

8:00 a.m.	10:00 a.m.	Council Breakfast/Meeting
10:00 a.m.	12:00 noon	House of Delegates Meeting
12:00 noon	1:30 p.m.	Opening Ceremonies/Lunch with speaker
1:30 p.m.	5:30 p.m.	Continuing Medical Education (4 hours)
7:00 p.m.	9:00 p.m.	Reception for Exhibitors/Physicians

Friday - June 8, 1990

7:30 a.m.	8:30 a.m.	Specialty Breakfast
9:00 a.m.	12:00 noon	Continuing Medical Education (3 hours)
12:00 noon	1:30 p.m.	Lunch with speaker
1:30 p.m.	5:30 p.m.	Continuing Medical Education (4 hours)
6:30 p.m.		Riverboat Discovery with Dinner

Saturday - June 9, 1990

6:30 a.m.		AAFP "Fun Run"
8:30 a.m.	11:30 a.m.	House of Delegates
11:30 a.m.	1:30 p.m.	Lunch with gubernatorial candidates
1:30 p.m.	5:00 p.m.	Continuing Medical Education (3.5 hours)
6:30 p.m.		President's Banquet/Dance

Sunday - June 10, 1990

7:30 a.m.	10:00 a.m.	Council Meeting Breakfast

REGISTRATION FORM

NAME: DATE:
ADDRESS:
ZIP:
NUMBER OF ADULTS IN PARTY:
SPOUSE'S NAME:
NAMES & AGES OF CHILDREN: AGE:
(in attendance)

HOW AND WHEN ARRIVING:
Send check or money order with Registration Form to: **Alaska State Medical Association**
4107 Laurel Street, Anchorage, AK 99508

Health 90

HEALTH PROMOTION CONFERENCE SUMMARY REPORT

In April, 1987 the Health 90 Conference occurred focusing on health promotion within the state. Funded by the Alaska State Legislature, a conference creating a Health Promotion Plan for the state was held. Nearly 300 people were involved in planning the meeting of 109 attendees representing 21 communities.

THE PROCESS

Health 90 was based on the report by the Surgeon General's office *1990 Health Objectives for the Nation*. As an effort to evaluate the state and serve as a change agent for the future, a systems approach was selected. Further, it was concluded that individual decisions to alter behavior were best supported when the systems around us promote health and healthy life-style choices. Five "systems" were identified as primary to this effort: the community, the educational system, the media, the medical system and the work site. Later, members of the media task force elected to withdraw "media" as a system since the function of the media was to "reflect public policy and not create it."

Promotion mailings were sent to 4,000 health organizations, private and public schools, churches, legislators, native corporations, villages, municipal and borough administrators, and state offices. Additionally, flyers were included in the statewide mailings of various organizations and associations. The media was contacted, presentations were made to small groups, and 3500 businesses were asked for involvement and support.

Key informants, representing groups with large or statewide constituencies, convened in Anchorage to assess the fifteen topic areas addressed in *1990 Health Objectives for the Nation.* Each topic area was evaluated to determine which of the four system approaches might most effectively impact behavior. These priority areas were then assigned to each of four task forces to evaluate, discuss and localize according to the Alaska Policy Recommendations.

Task forces were formed for each of the four systems, community, education, medical and work site; and statewide teleconferences were held. Objectives were prioritized for accomplishment by 1990. Normally, in most large scale planning efforts, the work would stop here. The goal of the conference planners was to prepare participants and policy makers with actual strategies enabling the state to reach the desired objectives by the year 1990. With focus and monitoring a three year baseline will provide data appropriate to planning, resource allocation, and service delivery during the nineties.

Following the conference, ideas were submitted to experts for review and comment. The final draft was presented to participants and interested parties with the opportunity to prioritize each into a high, medium or low category. The form return rate was 20 percent.

AWARDS

Awards were presented to a firm or individual for their efforts in promoting health and wellness in Alaska.

The Community Award was presented to Carrs Quality Centers for their "Permanent Fitness Dividend" project and to Yukon-Kuskokwim Health Corporation for their nutritional awareness project in the Bethel area.

Carolyn Peter, Education Program Designer and Training Director for the Alaska Counsel on Prevention of Alcohol and Drug Abuse received the Education Award for introducing and adapting the "Here's Looking at You" program in Alaska.

The Medical System award recipient was Ann Morris, M.D. Dr. Morris was one of the founders of the Healthy Alaska Coalition which lobbied successfully for the cigarette excise tax, as well as a participant in the early planning of the Health 90 Conference.

The Work Site Award was presented to Providence Hospital for their "Health Style" employee incentive program.

THE RESULTS

At the end of the conference, over 1000 rural and urban strategies resulted in moving closer to the goals by the year 1990. These strategies were stated in a general manner to enable communities, employers and professionals to create specialized activities locally appropriate. These strategies were grouped into three distinct categories: data, policy and education/service delivery.

PRIORITY DATA STRATEGIES

- Establish a centralized data collection site and "ongoing studies" registry.

- Monitor taxes raised on tobacco and substance sales and designate the entire amount for health promotion, education, and intervention.
- Establish and monitor an Alaska-specific Health Risk appraisal.

PRIORITY POLICY STRATEGIES

- Increase taxes on tobacco products.
- Require schools, medical and central offices, and work sites to be smoke-free and ban cigarette machines from these sites.
- Increase the legal age to purchase tobacco products to 21 and enforce "No Sale to Minors."
- Require mandatory substance abuse treatment for offenders and parents of minor offenders.
- Increase taxes on alcohol products.
- Increase fines and conviction rate for selling alcohol and other substances to minors.
- Establish "No drinking during workday" policies.
- Establish bike routes and recreation areas in all communities.
- Require school lunch programs to reduce sugar, salt and fat content.
- Include nutritious food choices in school and work site vending machines.
- Require all sexually transmitted diseases be reportable and strengthen reporting laws.
- Provide access to condoms in public restrooms for both sexes.
- Strengthen or develop new strategies to protect client health record confidentiality.
- Insure confidential family planning services for teens and adults.
- Restore funding for public health nurses.
- Maintain and enforce 55 m.p.h. speed limit.
- Support insurance differential for seat belt use.
- Establish latch key programs.
- Require and enforce lifejacket use and floatcoat or floatsuit access on all boats.
- Enforce alcohol ban on boats.
- Support change in federal ICD.9 codes to identify method of death-causing assault.
- Provide safe shelters or homes in all communities.
- Provide 24 hour crisis counseling.
- Require rigid enforcement of restraining orders.
- Strengthen parole monitoring of violent offenders.
- Establish company policies against verbal abuse and violent behavior on the job.
- Enforce law requiring employers to provide safety gear, equipment, and emergency information, then require employees to utilize the gear.
- Manage hazardous waste sites.
- Inspect, monitor, and enforce regulations against toxic water and leakage pollution.

PRIORITY EDUCATION AND SERVICE DELIVERY STRATEGIES

- Promote community health fairs and wellness projects.
- Utilize elders and community leaders as educators.
- Utilize public service announcements and print media for information.
- Teach refusal and coping skills and promote self-esteem utilizing established, successful programs.
- Require K-12 curriculum in substance abuse, safety, and infectious disease; additionally, sex education must be taught in 4th through 12th grades.
- Promote "cessation" programs and employee assistance programs.
- Promote local self help groups.
- Utilize community schools and establish "equipment lending" program.
- Promote physical activity for the handicapped and elderly.
- Train health professionals in nutrition, sexually transmitted diseases, risk factor identification, and criminal assault assessment.
- Research subsistence and local food values.
- Promote "safer sex" advertising and public service announcements.
- Promote prenatal care, parenting education and services.
- Establish community infant car seat loaner programs.
- Post "911" emergency number, crisis line number and poison control number on all public phones.
- Promote drivers education, boating/ATU safety and gun safety programs.
- Support substance treatment programs.
- Support intervention programs for abusers and victims.
- Train vocational education students and all workers on safety practices and emergency procedures.
- Provide technical assistance to businesses.
- Inform residents on hazardous and toxic wastes.

DISCUSSION

From the conference organizers' point of view, two areas of significance are discernible. First, considerable effort was undertaken to ensure participation by rural Alaskans. A substantial portion of the budget (25%) was allotted for subsidized travel, and applications for subsidized travel were included with every mailer. All native corporations, city mayors, and managers were contacted, using key mailing lists. Three factors come to mind which may have caused the low response:

1. The application requirement for justification of subsidy requests by interested persons..
2. The subsidy limit initially set at $137.50.
3. Limited posting and circulation of information resulted in reduced opportunity for staff to request time off or financial assistance to attend.

The second area pertains to the volume of strategies presented by the conference attendees. Splitting the working groups into rural and urban brainstorming groups was well received. Conference planners and advisors felt that 5-10 strategies would be developed for each objective, but the participants often generated lists of 50 or more. Efforts to eliminate duplications were undertaken, and some editing was required; however ultimately, the strategies remained intact for the conference report mailing which included a prioritization rating sheet. Naturally, a higher response rate to the prioritization question was preferred. Fortunately, the respondents reflect an appropriate cross-section of the range of participants.

There was cause for concern among some reviewers that compared certain strategies to "gestapo tactics." For example, strategies which suggested students should report on smoking and substance use in the home were eliminated. Further, strategies which implied inappropriately that the education system, the medical system, or work site should assume total responsibility for individual health (e.g. employers should provide health club memberships for all employees and their families) were eliminated.

Occasionally, the strategies were overzealous and while well-meaning, began to infringe on human rights and civil liberties. While some where left in the overall document, these were seen as low priorities with the need for further review.

SUMMARY RECOMMENDATIONS:

1. It is important that this be seen as the beginning of an effort and not the end of one.
2. It is important that the priority areas addressed here be treated as such and that funding of services reflect this.
3. It is important that Alaska residents monitor the decisions of the administration and policy makers and encourage their use of the document.
4. It is important that consideration be given to a similar effort for the remaining four areas addressed in the Surgeon General's *1990 Health Objectives for the Nation.*
5. It is important that a comprehensive data system be the foremost issue confronting the 15th legislature during its 1988 session.
6. It is important that health promotion be established as a priority in the Department of Health and Social Services.

The success of the conference rests largely on the efforts of the facilitators, mentors, chairs, sponsors and supporters, and conference planners. The complete document is available from the Alaska State Medical Association, 4107 Laurel Street, Anchorage, Alaska 99508.

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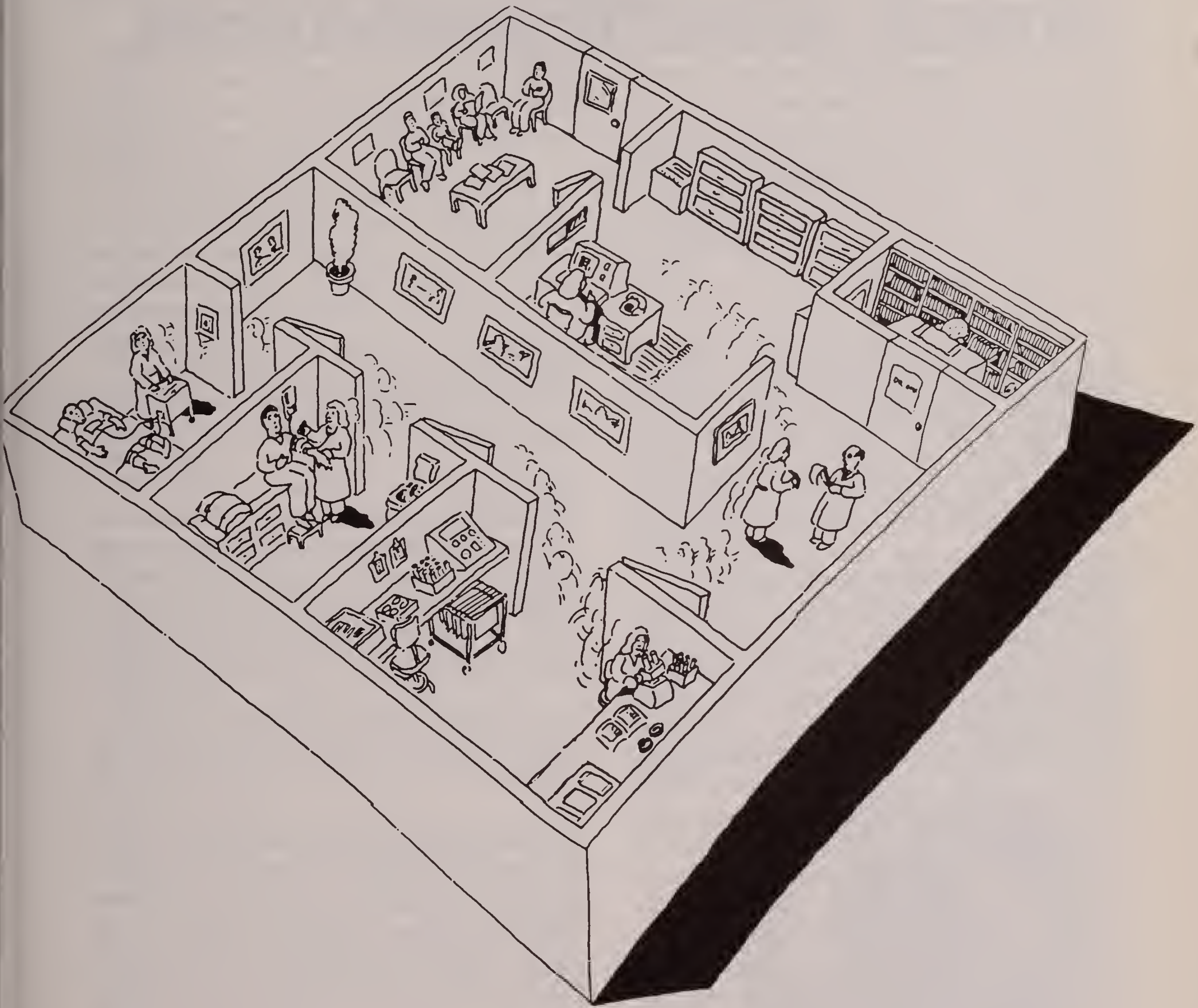

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ALASKA PUBLIC HEALTH ASSOCIATION

PRESIDENT'S COLUMN February, 1990

by Susan A. Jones, R.N., M.N.

In January the first "Task Force for Healthy Alaskans" meeting was held to explore the possible development of a state-wide health policy. Included at the meeting was the Director of Public Health, Katherine Kelley; Sally Mead from the Office of Prevention; Ray Schalow from the Health Care Coalition and your ALPHA president. The meeting was very energetic and endorsed the concept of "policy" development over "plan" development.

ALPHA is excited to see the growing fruits of its' labor in the commencement of this group. For the past three years ALPHA has been an advocate for the development of a state-wide health policy. Action by Commissioner Munson to begin the task force followed a letter that ALPHA sent in November. At that time ALPHA requested that Commissioner Munson establish a commission involving many public and private health care providers, and professional organizations. ALPHA interviewed Commissioner Munson in December and the idea of a smaller working group was born. The task group members share Commissioner Munson's hope that this policy development will continue through to the next administration and collect a broad base of support throughout the state.

Also in ALPHA's news is the establishment of the Alaska Health Fund. The purpose of this fund is to promote and support educational and professional growth activities for the enhancement of the members of the Alaskan health care community. Individuals and organizations engaged in health related activities within Alaska may request in writing for support. Although membership in ALPHA is not required, you do have to live in Alaska. Awards will be made for amounts less than or equal to $500.00 for individual awards and up to $1,000.00 for organization.

The total awards given are limited to 10% of the Alaska Health Fund. Ask board members for more information or see your January Newsletter for specific details.

ALPHA has received word from the Coalition for Injury Prevention and Control, APHA, that FY 1991 Coalition funding recommendations for CDC Injury Program will again recommend $50 million and 150 FTE's. In addition the group agreed that a major priority for the coming year will be the establishment of a "Center" for injury within CDC. One of ALPHA's goals continues to be focused on securing additional funds for injury control programs in Alaska.

The annual ALPHA business meeting and awards banquet is developing into an exciting event. Under the creative orchestration of President-elect Brian Saylor you may find the international flavor of the Circumpolar Health Congress reflected at the banquet table with representatives from USA and Canada. We'll have good food, good conversation and awards. Mark May 23 on your calendar for the annual meeting and awards banquet.

ALPHA had 171 members at the end of 1989. Renew your membership and have a friend join, we need your support both through activities and paid membership. We are involved in interesting and exciting activities and want you to share in the excitement.

DEATH AND DESTRUCTION
(and programs to deal with them)
by Carl Hild

"It was twenty years today, when Sergeant Pepper taught his band to play. It's been going in and out of style, but it's guaranteed to raise a smile. So may I introduce to you..." Earth Day twenty, ooh! It is also a time to remember those who have died on the job - Workers' Memorial Day.

That's right, back by popular demand is the ecology symbol of the yellow capital Theta with all of its significance for Earth Day. There will be programs throughout the country raising awareness for environmental health issues. ALPHA has passed resolutions in the past that support many environmental concerns. It is important to individual and public health that we all have clean air, clean water, clean food, and an aesthetic environment in which to live.

The past few years have been tough here in Alaska with water contamination in many areas of the state, air pollution exceeding federal guidelines in our largest cities particularly in the winter, and the closing of much of the fishing season due to the Exxon Valdez spill. Alaska is too small to waste. Many rural communities have serious trash disposal problems.

The new international marine pollution laws are putting port communities into the bind of having to receive wastes off vessels when they have little room for their own wastes.

There must be a new focus, and that is on waste reduction. Each and every one of us must stop producing so much waste. Earth Day activities on April 22, 1990 will have many suggestions on what each of us can and must do if we are to save "spaceship Earth" from our own wasteful ways. Earth Week - April 22-28, 1990.

One major form of waste reduction that we need to focus on is the waste of human health on the job. Workers' Memorial Day is the last day of Earth Week, April 28, 1990. This day has been selected as a day of mourning for those who have been injured or killed on the job. Nationally 15,000 will die doing their job this year and 100,000 more will be injured. Alaska ranks as one of the states where death or injury on the job is highest.

Workers' Memorial Day is not like the other Memorial Day when those who died defending our country are thanked and remembered. The goal of Workers' Memorial Day is to "fight for the living." It, too, was twenty years ago when the Occupational Safety and Health Administration was established to set standards for workplace safety. We all need to do something in our workplaces today, to reduce the number of deaths and injuries and to meet OSHA standards. We remember the dead so that we can work for the health of the employees of today.

ALPHA has in the past had resolutions supporting workplace safety and health programs. We need to be aware of Alaska's high rate of injury and death on the job and as health professionals we need to help others become more aware of this issue. Occupational health has been called the stepchild of public health. It is about time it becomes a full part of the family.

ALPHA members and all health professionals are encouraged to participate in local activities for both of these National events. Earth Day, April 22, and Workers' Memorial Day, April 28, are two chances to work actively in your community to reduce death and destruction. For more information on these programs contact Carl Hild, Alaska Health Project, 276-2864 or 800-478-2864.

ECOLOGY: 1. The branch of biology which treats the relations between organisms and their environment: Bionomics 2. The branch of sociology concerned with the spacing of people and institutions and their resulting interdependency. [Formed from modification of Greek (Oiko) House + logy] (Household)

IN OTHER NEWS!!

** The ALPHA board would like to congratulate Bruce and Jennifer Talbot on producing a potential new member through the birth of Cole Williams Talbot on January25, 1990. Jennifer Talbot has been an active board member and your newsletter editor.

** The Alaska Health Fair is 10 years old in 1990!

** The American Public Health Association (APHA) is asking us to join in applauding Dr. Louis Sullivan, Secretary of Health and Human Services for blasting the "R.J. Reynolds' proposed marketing of "Uptown" cigarettes, a brand blatantly targeted at the African-American community in Philadelphia." R.J. Reynolds cancelled the "Uptown's" premier. APHA requests you write to both Dr. Sullivan and President Bush to indicate your support for Dr. Sullivan's stand against "Uptown."

Louis Sullivan, M.D.
Secretary of Health
& Human Services
U.S. Dept. of H & HS
200 Independence Ave.
SW #615 F
Washington, D.C. 20201

The Honorable George Bush
President of the United States
The White House
1600 Pennsylvania Ave., NW
Washington, D.C. 20500

For more information, call Deborah McLellan, APHA, 202-789-5600.

** Interesting news from the DEC (Alaska Department of Environmental Conservation), they plan to continue a study of Nome women next year to determine if mercury contamination has resulted from offshore Norton Sound gold dredging. The DEC, along with the Federal Minerals Management Service, last summer began testing the hair of 200 Nome women of childbearing age to determine mercury levels. Findings revealed the mercury level was 1 part per million (ppm). The risk threshold from the Environmental Protection Agency for fetuses is 6 ppm.

**** ANNUAL MEETING REMINDER!! ****

** The Annual Meeting will be held in WHITEHORSE on MAY 23, at the General Store in the GOLD RUSH INN from 4:00-7:00 p.m.

** The Annual Banquet will be in the TOWN HALL of the Gold Rush immediately following the Annual Meeting. Cost for the banquet will be $25.00 (U.S. Funds) for ALPHA members and $30.00 (U.S.) for non-members. It is sure to be excellent, so get your reservations in early!!
RSVP to Jan Wills no later than MAY 1, 1990

Enclose payment and send to: Jan Wills
Division of Community Health Services
Dept. of Health and Human Services
Municipality of Anchorage
PO Box 196650
Anchorage, AK 99518-6650

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45th Annual Meeting

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EXHIBITORS: Booth space is only $500 for a large 8' x 8' size, assigned on a first pay basis. Exhibitors that support ASMA this year will have first bid at the convention next year in Magadan, USSR. Since it appears that space will be limited on our trip to Russia, only a limited number of exhibitors/attendees will be going.

RESERVATIONS: Hotels - Contact the Westmark Inn, The Super 8, Sophie's Station, or the Regency in Fairbanks. Exhibit Space - Call ASMA or send payment to:

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Program Notes
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INTERMEDIATE ANTIBIOTIC RESISTANCE OF INVASIVE STREPTOCOCCUS PNEUMONIAE ISOLATES RECOVERED FROM ALASKAN RESIDENTS

During 1986-1988 Statewide invasive pneumococcal disease rates for Alaska Native infants (under 2 years of age) and adults of various ages were 6 and up to 14 times higher respectively than non-Native rates, and 17 and up to 10 times higher respectively than rates reported for the U.S. general population. In the Yukon-Kuskokwim River Delta (YKD) during 1980-1986 infant rats were 34 times higher than other U.S. populations.

Penicillin remains the treatment of choice for invasive pneumococcal infections. However pneumococci are no longer uniformly susceptible to penicillin. Generally, 1-4% of clinical isolates recovered in the U.S. show intermediate penicillin resistance (IPR), also referred to as partial penicillin resistance or moderate penicillin susceptibility (IPR=MIC o.1-1.0ug penicillin/ml), and reports of partial and complete penicillin resistance and multiply resistant pneumococci are becoming more frequent. Where common in other countries, complete penicillin resistance has been preceded by partial resistance.

As part of Statewide surveillance of invasive pneumococcal disease, isolates were obtained from cultures of blood and CSF from Native patients admitted to the Yukon-Kuskokwim Delta Regional Hospital in Bethel, and the Alaska Native Medical Center in Anchorage, and from non-Native patients admitted to other Statewide hospitals. Isolates submitted were confirmed as S. pneumoniae by Optochin sensitivity and bile solubility. Serotyping was performed by Dr. Richard Facklam, CDC Atlanta.

Antibiotic minimum inhibitory concentration (MICs) were performed by the agar dilution method, described by the National Committee for Clinical Laboratory Standards (NCCLS), on 338 pneumococcal isolates recovered from normally sterile sites (blood, CSF) from Native and non-Native patients admitted to the YKD Regional Hospital (YKDRH) and Anchorage area hospitals between January 1, 1980 and through December 1, 1988 and statewide between January 1, 1986 through December 31, 1989. Of 110 Native patients from the YKDRH, 14 isolates (12.7%), showed IPR with MICs of between 0.1-1.0ug penicillin/ml. Ten (74.1%) had MICs of 0.12ug per ml and 4(28.5%) had MICs of 0.25ug per/ml. Twelve were from infants <24 months of age. In this region the annual rate of IPR varied from 5.2% to 26.6% (Table 1). Serotypes that were IPR were 6B, 14, 18C, 19A, and 23F with 54.4% of type 6B and 50% of 19A isolates demonstrating IPR. All other 19A isolates had MICs only one dilution lower. In contrast none of 66 isolates recovered from Native patients in Anchorage were e IPR and only 2 of 162 (1.2%) isolates recovered from non-Native patients from the Anchorage area hospitals were IPR. In all areas, for serious non CNS pneumococcal infections, moderate to high dose penicillin remains the antibiotic of choice since no isolates have been identified in Alaska that are not readily susceptible to achievable serum levels.

Table 1

Intermediate Penicillin and Chloramphenicol Resistance among Blood and CSF Pneumococcal Isolates from Native Patients from the Yukon-Kuskokwim Delta Regional Hospital
1982-1988

Year	Total Isolates	*IPR (%)	**ICR (%)
1982	4	1(25.0)	0(0)
1983	22	3(13.6)	2(9.0)
1984	23	3(13.0)	4(17.3)
1985	19	1(5.2)	6(31.5)
1986	11	1(9.0)	4(36.3)
1987	15	4(26.6)	3(20.0)
1988	15	1(6.6)	0(0)
Total	110	14(12.7)	19(17.2)

*IPR=intermediate penicillin resistance o.1ug-1.0ug penicillin/ml.
**ICR=Intermediate chloramphenicol resistance 4.0ug chloramphenicol/ml.

Intermediate resistance to chloramphenicol (ICR), defined by a MIC=4.0ug/ml, was found in 17.2% of isolates from Native patients from the YKDRH. Similar rates were detected in isolates from both Native (16.6%) and non-Native (15.4%) patients from the Anchorage area hospitals and elsewhere in Alaska. Overall 52.9% of type 9V and 47.3% of type 12F were ICR. Chloramphenicol is frequently used in combination with penicillin for CNS pneumoccal infections, in patients with penicillin hypersensitivities, or in CNS infections of insensitivity to penicillin. Because pneumococcal isolates with MICs of 8.0ug/ml chloramphenicol/ml are classified as resistant (the average serum level with normal dosage patterns), isolates that are ICR can be used as potential indicator of chloramphenicol resistance. Despite higher achievable CSF levels of chloramphenicol relative to serum levels (66:1) compared to penicillin (3-5:1), clinicians may wish to consider a third generation cephalosporin (CSF:serum=4-16:1) for initial treatment of gram positive coccal meningitis in Alaska Native patients from areas with proven IPR such as the Yukon-Kuskokwim Delta until antimicrobial sensitivities are available.

Although case/fatality ratios for Alaska Natives with pneumococcal disease have not been higher than expected, of 11 deaths among Native patients from whom pneumococcal isolates were recovered, two patients had pneumococcal isolates that were IPR With MICs of 0.25ug penicillin/ml, and one patient had an isolate that was ICR. This underscores the need to screen all pneumococcal isolates recovered from normally sterile sites for penicillin and chloramphenicol susceptibility, particularly CSF isolates. Penicillin susceptibility can be estimated overnight by disc diffusion using either a lug oxacillin disc rather than a penicillin disc and chloramphenicol susceptibility by use of a 30ug chloramphenicol disc. A bacterial suspension approximating a 0.5 McFarlane standard should be used to inoculate blood agar. The following zone sizes can be used to estimate susceptibility:

	susceptible	intermediate	resistant
oxacillin	≥20mm	13-19mm	<13mm
chloramphenicol	≥18mm	13-17mm	<12mm

Among all invasive isolates tested, from both regions of Alaska, two isolates were resistant to tetracycline, and one other was resistant erythromycin. Intermediate resistance to tetracycline was found in two isolates, to erythromycin in two, and to ampicillin in two isolates. No multiply resistant or fully resistant strains of S. pneumoniae were found in this survey. All isolates were susceptible to trimethoprim/sulfamethoxazole, ceftriaxone, cefotaxime, cephalothin, cefaclor, and vancomycin.

The Arctic Investigations Laboratory together with 23 participating hospital laboratories and clinics continues to monitor invasive pneumococcal disease occurring throughout Alaska. This laboratory based system of surveillance requests that any pneumococcal isolate recovered from a normally sterile site (blood, CSF, pleural space, joint) be sent to AIL. Isolates are serotyped and screened for antimicrobial susceptibility. this information is available to participating laboratories. This surveillance provides critical information regarding disease incidence necessary for recommendations on antimicrobial therapy and disease prevention.

Direct any inquiries regarding this surveillance program to Dr. Alan Parkinson, (907) 271-4011.

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NEWS FROM THE AMERICAN SOCIETY FOR CIRCUMPOLAR HEALTH

At the close of the VIIIth ICCH in Whitehorse, our President, John Middaugh, will be ending his tenure and the President-Elect, Anne Lanier, will begin her term of office. We are all deeply grateful to Dr. Middaugh for the vision and inspiration, energized dedication, leadership and many accomplishments he has provided to the Society. His guidance will be missed, but we look forward to his continued activity with ASCH.

Dr. Anne Lanier has also been a member of ASCH since its inception and a two term Board member. We look forward to her term as President and working together.

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TRAVEL INFORMATION TO WHITEHORSE:

Transportation to Whitehorse from the Southcentral area of Alaska will be either via air or driving down on the Alaska highway. The highway bus system will not be in operation until after June 1st, and chartered bus rates are too high. There are regular scheduled flights to Whitehorse via Fairbanks or Juneau on Fridays and Saturdays. These involve connecting flights from Anchorage and approximate fare will be $425 (RT).

Special Charter Flights from Anchorage direct to Whitehorse on May 19 and May 20 with return on May 25 have been arranged with Delta Air Charter Ltd. of Whitehorse, Y.T. The flight time will be approximately 3 hours and total cost will be $385 (US).

Space is limited as the plane capacity is 18 passengers. This is the best transportation price to Whitehorse so if one wishes to utilize the chartered flights, it is recommended reservations are made as soon as possible.

FOR FURTHER INFORMATION CONTACT:

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Phone: 907-279-7461

For those driving or wishing to camp, there are RV parks and tenting areas scattered around the outskirts of Whitehorse. There is also an RV park in town directly across from the Westmark-Klondike Inn with full hook-up.

FOR MORE INFORMATION, MAPS, ETC, CONTACT:

Sourdough City RV Park
PO Box 4761
Whitehorse, Yukon YIA 3TC
Phone: 403-688-7938

Whitehorse Chamber of Commerce
302 YTG Steel St.
Whitehorse, Yukon, CANADA
YIA 2C5
Phone: 403-667-7545

Get Acquainted Breakfast

ASCH is planning a no-host get acquainted buffet breakfast for Society members and all U.S. registrants participating in the VIIIth ICCH. This is a good chance for everyone to get acquainted and learn more about ASCH and its activities.

The buffet breakfast will be from 7 - 9 a.m. on Thursday, May 24, 1990 at the Westmark-Klondike Inn.

Price will be approximately $9 - 10 (Can.) with full details at registration. A special bus will be available at the end of the breakfast to take all attendees to the ICCH plenary session at Yukon College.

To help the committee in planning the breakfast, please complete and return the attached form if you are attending the VIIIth ICCH and plan to attend the breakfast.

[ ] I plan to attend the breakfast on May 24, 1990, 7 - 9 a.m., Westmark Klondike Inn

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History of Medicine in Alaska
William Harvey Ivy, M.D.

The son of parents, who both earned PhDs in physiology, Bill was named William Harvey after the great 17th century physiologist who discovered the circulation of blood.

His mother, Emma Kohman, was born on a homestead in central Kansas, one of thirteen children. She proved that nutritional edema was caused by lack of protein in the diet and in the process helped establish the value of animal experiments in the study of human disease. When she married Andrew Conway Ivy in 1919, she chose to become a homemaker only returning to the laboratory after her five sons, four of whom became physicians, were grown.

His father taught physiology at the University of Chicago and Loyola Medical School which enabled him to attend Rush Medical School from which he received an M.D. He then began a long association with the Northwestern University School of Medicine. He enjoyed teaching, feeling that "it is the duty of those who know to teach". Despite a varied career doing research, writing papers, and holding high academic positions, some of them simultaneously, Dr. Ivy considered the training of physiologists and gastroenterologists his greatest contribution.

William Harvey was to be both an excellent student and a fine athlete. He was a member of the National Honor Society in high school and was graduated Phi Beta Kappa from Northwestern University. Also he was a heavyweight wrestling champion in the Chicago high schools in 1939 and captain of both the Bowen High School football and wrestling teams.

His football career was complicated. He played as an undergraduate for two years at Northwestern and was honored to play left tackle in the East-West Bowl in 1943. However, when serving in the military in the Army Specialized Training Program (ASTP), he was not allowed to play football.

After release from the Army, he played a third year of football while a senior at Northwestern Medical School. He accomplished this by not paying his graduation fee and thus not officially graduating from the undergraduate school. In 1946, while a medical student, he played in the North-South Bowl. The same year, he was named All-American Tackle, Northwestern Big Ten. Bill Ivy was drafted by the Philadelphia Eagles and the Washington Redskins. This became a moment of truth when he had to decide between medicine and football.

While World War II had shortened both college and medical school by working all year around completing four years in three, even this was interrupted for Bill Ivy. He chose to go on active duty, dreaming of going to Europe and driving a tank. World War II was the last crusade in which active involvement in fighting was sought. Of course, when his medical school background was discovered, he was sent as a corpsman to Brooks Army Hospital in San Antonio to administer penicillin shots in peanut oil to VD patients. This was so boring that he volunteered for duty escorting discharged violent psychotic patients back to their local VA psychiatric institutions. He traveled around the United States by Pullman using his wrestling techniques to subdue the uncontrollable.

It was not hard after medical school to plot his course. Pay in professional football wasn't too promising in those days and he was drawn to medicine. He took an internship at King County Hospital in Seattle where pay was negligible. However, there was the bonus of meeting a young nursing student, Geraldine Lough, in July and marrying her in September 1947. They had an apartment near the hospital where they set up housekeeping. This enforced life-style of constant accommodation of conflicting schedules somehow promoted an enduring forty-two year relationship.

Bill practiced for a year in a clinic in Raymond, Washington, but he was constantly urged by his friend, Vern Cates, to come to the Doctors' Clinic in Anchorage, where doctors were scarce. He was the twelfth physician in town.

Dr. Harold Sogn, who founded the clinic and had a large obstetrical practice, suffered a heart attack. Bill Ivy found himself covering this practice. Eventually, Dr. Sogn recovered and after returning to his practice, invited Dr. Peter Koeniger to Anchorage.

At this point in 1955, William Harvey Ivy was redrafted during the Korean War on the technicality that he had not served twenty-four months in the army because his time in the ASTP was not counted. Again his hopes of serving abroad were dashed. He was stationed at Elmendorf Air Force Base. He was assigned to the obstetrics department where there were many board qualified obstetricians so that the two years were equivalent to a residency in the field into which he had fallen by chance.

In spite of a large Ob-Gyn practice, he had some loyal General Practice patients including some men who often found themselves greeted by the office staff with "have you had your Pap smear lately?"

In his early years of practice in Spenard, he encountered a typhoid outbreak which was started by a carrier who had baked cookies for the Boy Scouts. He also identified one case of Parrot Fever (psittacosis) caused by a parakeet.

Bill speaks with a combination of amusement and consternation about an early confrontation between sanitation and the Anchorage Ski Club. The latter, having established a ski area at Arctic Valley, took the usual Alaskan attitude that outhouses were not a hazard in such a large and relatively unpopulated area. The observant physician realized that this was indeed a potential public health problem because the ski area was located in the Ship Creek watershed from which the Anchorage water supply came. While the military treated its water, the city did not and occasionally unusual items popped out of faucets.

Correcting the situation was not an easy battle because the understaffed health department left the problem to Dr. Ivy who found himself battling the entire militant ski club headed by the ebullient Muktuk Marston.

Public arguments about zoning and sanitation which were common happenings thirty years ago have long since disappeared from Anchorage. These were Town Hall meetings with strong emotions. Consequently, the first effort ended in a debate at the Fourth Avenue Theater between Muktuk and Bill. Marston took the winning stand that if moose can defecate on the tundra, why can't man? Bill was persistent however and Arctic Valley eventually installed appropriate facilities.

In those days there were no emergency rooms. Thus many patients were met on night house calls. Because it was often very dark with no street lighting, it was difficult to locate houses. Directions were often obscure - e.g. "Drive 1.2 miles up the road" or "the sixth house on the right surrounded by spruce trees".

Most fascinating were two cases of acute glomerulonephitis which were traced to Beta hemolytic strep in a school bus. Bill remembers the house call to the hillside which was probably O'Malley Road. He drove on a gravel road toward Potter where a rendezvous with a weapons carrier provided transportation uphill to a cabin. There were two very sick children, one with a puffy face. Bill determined the diagnosis by boiling urine in a spoon over the stove as he was taught in medical school. The boys were taken to the hospital where one died. All of the school bus occupants were treated prophylactically with penicillin.

But the high incidence of tuberculosis upset him the most. He had OB patients with TB. All that was done at the time was to put their names on a list and when the name came to the top they were sent to the Seward Sanitarium. Bill read an article in a Michigan journal about effective outpatient treatment of TB. It advocated streptomycin, monitoring and education.

He went to the Board of Health to protest tuberculosis care. This lead to his appointment to the Greater Anchorage Health District Board of Health. He later became chairman.

Urged on by the Board of Health, Bob Wilkins, M.D.; Merritt Starr, M.D. and Francis Phillips, M.D. set up and staffed an Anchorage based outpatient treatment center. There was much education involved in the program, and the evaluation of contacts helped to contain the disease. This was done with the full cooperation of the Public Health Department which was headed by Dr. John Gentry, USPHS, and some wonderful participation by some PHS nurses.

Since in the early 1950s there was no ambulance, a hearse was used for this purpose. The first ambulance, dubbed "Little Red Wagon", was to come almost ten years later. Also, since there was no municipal morgue, autopsies in Anchorage were and are still performed in mortuaries.

Bill has strong memories of the polio epidemic of 1953. Some of his patients were victims. He recalls Helen Whaley, M.D. and John Tower, M.D. working steadfastly. There were problems of not knowing how much oxygen to give to iron lung patients and how to avoid carbon dioxide intoxication.

Later, Bill was to be involved locally in the national controversy about Krebiozen, a drug thought to be a stimulator of the immune system and useful in the treatment of cancer.

When a group of Anchorage physicians recognized a need to establish another full service medical center in the city, Bill Ivy was extremely active. The effort began with the building of the Presbyterian Community Hospital on 8th Avenue and L Street in 1960-63. The facility grew rapidly and later its name was change to Anchorage Community Hospital, still later to Alaska Hospital and Medical Center and finally it was sold to Humana, Inc. Bill devoted time and energy to the development of the facilities, the organization of the medical center, and especially recruitment of personnel. He was chief of staff for five consecutive years. He feels that this was his greatest contribution to the health care delivery system of the Anchorage area.

He is somewhat disenchanted with current medicine. He is shocked at the cost of a C-section, feeling that people cannot afford medical expenses such as these. There is too much government sponsored waste as well as the sorrow of what malpractice suits have done to the system. He notes, for example, that medical schools no longer teach forceps techniques and some other alternatives to C-sections.

None of the Ivy offspring went to medical school, however two daughters are nurses. Their three other children have interests evolving from an early exposure to building and carpentry. One daughter is an interior designer; one son an architect; another an architectural project manager. The Ivys had another son who died in infancy.

Gerrie Ivy has had a long and varied volunteer career. Much of this has been with the American Red Cross. She started out in Water Safety/Lifesaving, teaching at first in the public school system. Then with the encouragement of Helen Dittman Beirne, Gerrie became involved in the Adaptive Aquatic Program for the handicapped which was established at the Alaska Treatment Center. Having begun this in Anchorage, she took the program to Idaho, Massachusetts and Hawaii. Later she taught children with orthopedic problems in her own pool.

She has served on the board of the Red Cross and as its chairman.

In 1972 she became an instructor in the Associate Degree nursing program at the University of Alaska, Anchorage. She served for fourteen years full time and continues to teach part time.

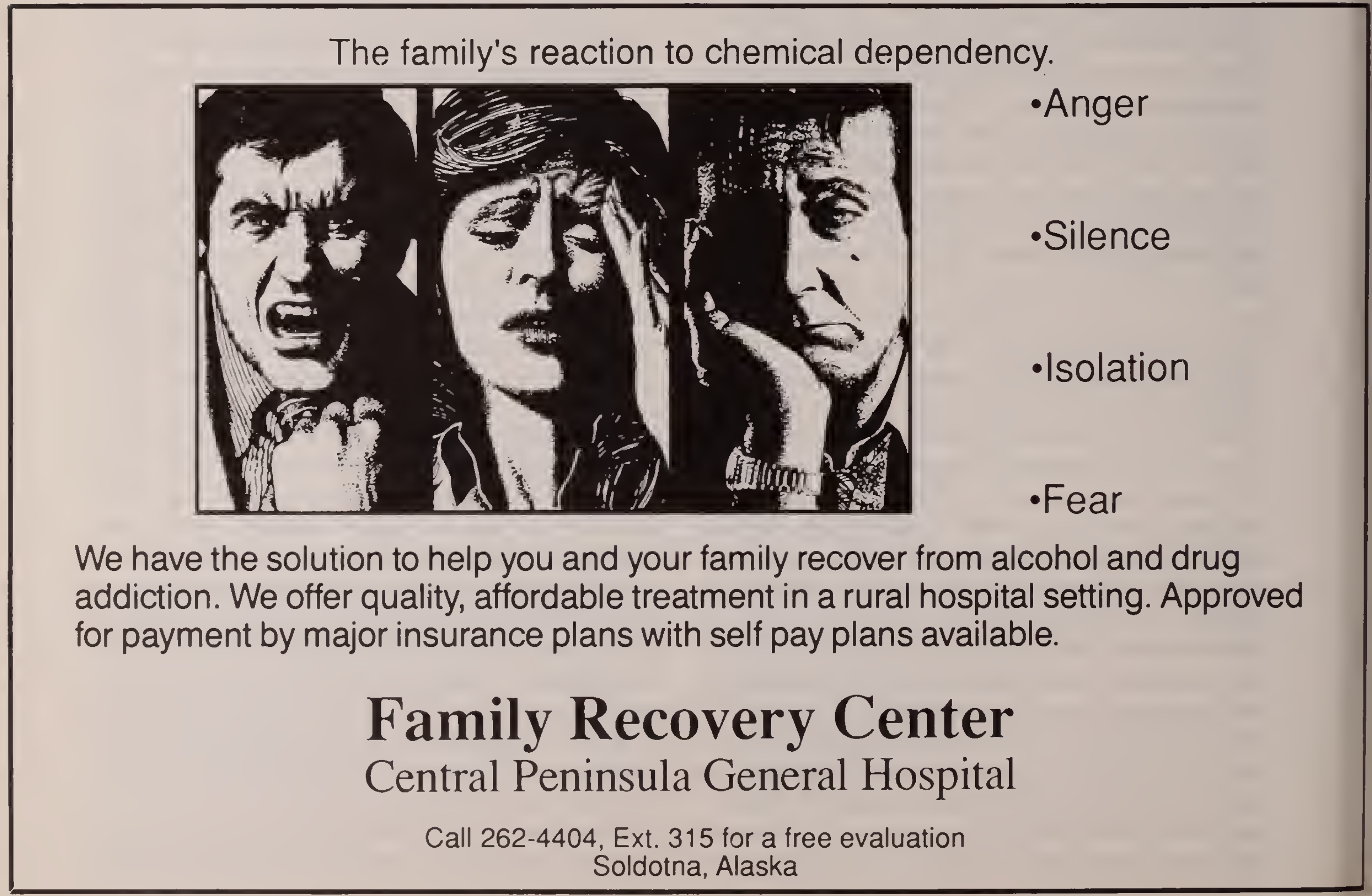
Bill and Gerrie helped start the Alaska Sailing Club on Big Lake, teaching basic sailing.

Both the Ivys have licenses to fly single engine planes on skis, wheels and floats. They have used these skills to explore Alaska. They have landed between the Diomedes, on Mt. McKinley glaciers, and in the Brooks Range. They have considered visits to native villages outstanding experiences. Gerrie and Bill have collected many fascinating artifacts: harpoons, ulus, usicks, masks and baskets, some of these were found as they hiked. They have chosen a section of Alaska to explore nearly annually and have seen most of the state including the Alaska Peninsula.

They have homesteaded where Bill enjoyed building cabins. Gerrie claims his children saw much more of him because he did this. They saw him more as a carpenter than a doctor and after much home health care, one even asked: "May I go to a real doctor?"

Bill and Gerrie represent the best of Alaska: professional, involved, developing skills to know and live in Alaska, caring about the Great Land and its people.

Gwynneth Gminder Wilson
Alaska State Medical Association Auxiliary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GLIMPSES OF ALASKAN MEDICAL HISTORY

Edited by Robert Fortuine, M.D.

A WORKER'S LIFE AT NEW ARCHANGEL (1805-6)

Aleksandr Baranov built the first Russian settlement in southeastern Alaska in 1799, only to have it destroyed by the Tlingit three years later. In the fall of 1804 he returned with a large force of Aleuts and Koniag, and, aided by the men and cannons of the Russian warship *Neva*, was able to defeat a determined force of Tlingit warriors. The settlement he founded and named New Archangel is modern-day Sitka.

The following summer Count Nikolai Rezanov, ambassador of the tsar, visited the colony while on an inspection tour of Russian-American Company trading posts. Accompanying him was Dr. Georg H. von Langsdorff, a German physician who had served for a time as naturalist on Kruzenshtern's famous voyage on the *Nadezhda*. Langsdorff was a learned man who was inclined to be overly impressed with his own erudition, yet it is clear from his writings that he had a feeling for social injustice.

Rezanov's party decided to spend the winter at Sitka, thereby putting considerable strain on the food stores of the fledgling colony. Within a few months scurvy had broken out among the workmen, who were kept under abominable conditions. Food was plentiful at first, but nearly everything of antiscorbutic value, including fresh meat and fish, was reserved for the officers' table. By the end of January eight had died and about 60 were too sick to work. Health conditions in the barracks continued to deteriorate until finally in early March a ship was sent, with Rezanov and Langsdorff aboard, to the Spanish colony at San Francisco. By the time it had returned in June, however, most of the sick were already on the mend, as a result of a good herring run and the appearance of fresh plant growth. A total of seventeen Russians had perished during the winter, not to mention an unknown number of Aleuts, Koniag, and Creoles who were forced to eat the same food as the workers.

Langsdorff's ideas on the etiology of scurvy, in the account that follows, were widely accepted in his day.

"Many of these needy and diseased beings, who were kept daily to very hard work, were unfortunately in debt to the Company, and it not infrequently happened, that when wholly exhausted, and lying on a sick-bed, they were driven to their work with blows. The consequence is obvious: they sunk one after another, wholly exhausted, a prey to the scurvy, and all work was in danger of being stopped. . . .

"In the month of February, out of a hundred and fifty of the youngest and most healthy men that had been selected from the different settlements and brought hither, eight were already dead, and more than sixty were laid up in the barracks with their strength wholly exhausted, and full of scorbutic sores; the chambers in which they lay had neither stove nor chimney, and the windows were shut close and nailed down. The rooms were only warmed by the pestilential breath of such numbers huddled together; and to crown all, not the remotest idea of cleanliness prevailed among them....

"The scurvy commonly shewed itself first by debility, listlessness, and melancholy; inflammatory spots, sometimes larger, sometimes smaller, then appeared on the legs from the knees to the toes, which in a short time turned to sores. Those who were thus afflicted were not required to work, but were set to mount guard day and night in the cold and wet: this was alleged to be necessary for the public security: for the love of their native country these poor wretches were doomed to die in misery. It was a commonly received opinion that exercise was very salutary in the scurvy; the weakest among the sick were therefore dragged about by their comrades; and others, who had still some strength left, were made to draw or carry heavy stones about the room. . . .

"Considering it my duty as a physician, I endeavoured to give the sufferers every assistance in my power; but my voice . . . was too weak to be heard. In vain did I entreat to have a dry, warm, airy, and clean apartment allotted to the sick, and pointed out a place where they would be tolerably well situated; in vain did I desire to have all the medical assistance afforded me that circumstances would permit. . . .

"Better food was as little to be obtained for the sufferers as a better lodging. Very often when I applied to the overseers for this purpose, representing that sugar, rice, molasses, and other wholesome kinds of nourishment, were of more importance to the sick than medicine, and that a wholesome liquor might be made for them to drink from the spruce-fir with molasses, they laughed at me, and said, 'that must be a pretty doctor who would cure his patients with good eating and drinking, instead of medicine.'

REFERENCE

Langsdorff, Georg H. von. Voyages and travels in various parts of the world during the years 1803, 1804, 1805, 1806, and 1807. Two vol. [1814]. Reprint. New York: Da Capo Press, 1968. Vol. 2, pp 93-97.

President's Corner

Happy to report that the Magadan convention plan is still on track. During a recent meeting with Dr. A. Lebedev, the Magadan area health care administrator, a suggestion was made by him to use a cruise ship as a hotel since Magadan lacks hotels and restaurants. We may need to bring our tents, and to brown-bag it, but we hope to provide suitable nautical accommodations.

Back to the language --. In the ninth century, a Greek Orthodox priest named "Cyril" was dispatched to minister unto the locals in the northern wilderness. He was to the Slavs as the nineteenth century Christian missionaries were to the Hawaiians -- no written language existed so he invented a phonetic alphabet, appropriately thereafter called the Cyrillic alphabet. Since he was Greek, it's not surprising that he based his creation on his own phonetics, thus the use of alpha, gamma, delta, kappa, lambda, pi, rho, phi and perhaps a few others. For sounds with no Greek equivalent, he invented symbols such as the tseh, cheh, shah, shchah, etc. Unexplained is why a "beta" became a "veh", but happily a lower case beta with a little lean-to roof became "beh", so it's still recognizable. Also unexplained is why C became ess instead of sigma. Since the Russian word for "Union" is "Soyuz", USSR becomes SSSR which translates to CCCP. Russian grammar is very complicated. Approximately the last third of most words is a grammatical ending and can be ignored when you're trying to read. So as I said last issue, learn the alphabet and you will be surprised at how much you understand.

For his efforts, ol' Cyril achieved sainthood, presumably posthumously.

Donald R. Rogers, M.D.
President
Alaska State Medical Association

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Auxiliary News

ALASKA STATE MEDICAL ASSOCIATION AUXILIARY

Auxiliary has been busy with its annual projects this fall and winter, the first being the AMA-ERF fund raising project, our Holiday Sharing Card, which raised $2810. Money raised through the Sharing Card project is sent to the National Auxiliary and then dispersed to the medical school of the contributor's choice. Nationally the AMA-ERF makes contributions of more than $2 million yearly, a visible sign of medicine's continuing commitment to excellence. Thank you to all who helped with the project and participated in this very important fund raising effort.

Our scholarship committee is currently accepting applications for the 1990 Alaska State Medical Association Auxiliary scholarship awarded each spring. Two $1,000 scholarships will be awarded.

Plans are underway for an auxiliary program on relationships. Per request our program will address issues suggested by members directly related to being a physician spouse. Information and details will be mailed to home addresses under separate cover.

Once again auxiliary will be actively involved in the Tort Reform movement. Anchorage area auxilians interested in getting involved may contact me directly (before I call you). those of you in other areas should contact your local medical society office. Auxiliary was a large force in last year's tort reform efforts and we need to keep up the good work.

It's not too early to begin planning your trip to Fairbanks in June for the state convention. Auxiliary will have its annual meeting with installation of new officers. There will be informal educational programs planned for auxilians throughout the convention as well as a few fun afternoon adventures.

Lindsay A. Hansen Tucker
President, ASMA Auxiliary

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Guest Editorial

POLAR REGIONS - THE DECADE TO COME

by Peter E. Wilkniss, Ph.D (1)

The decade of the 1990s is imminent. It bears examining the question: "Is the community, which is dedicated to research in the polar regions, adequately prepared for the new decade and the beginning of the next century?"

It is obvious that in the last few years the polar regions, and their related research, have emerged from their former isolation. Highly publicized examples with political implications attest to this observation. To wit: the Antarctic ozone Hole, perturbed Arctic atmospheric chemistry, and the Montreal Protocol on Chlorofluorocarbons; the natural resource issues, such as ANWR in the North, and the Antarctic Minerals treaty in the south; and similar problems resulting from ship accidents and oil spills, like the Bahia Paraiso at Palmer Station in the Antarctic peninsula, and Valdez in Alaska. In all of these situations, timely credible results from scientific research are imperative in providing for informed, political decision-making.

Activities with potentially overriding influence on scientific programs and, consequently, on the conduct of research itself, have intensified. A number of legislative proposals concerning environmental issues, including the polar regions, are being introduced in the U.S. Congress. Under the Executive Office of the President, the Office of Management and Budget is scrutinizing programs and expenditures on global environmental change programs across the board of governmental departments and agencies, with the key, supporting role being played by the interagency Committee on Earth Sciences. The Scientific Committee on Antarctic Research, part of the International Council of Scientific Unions, has introduced initiatives to link Antarctic research to the Global Change/International Geosphere Biosphere Programs. In the North, the Arctic Nations are engaged in encompassing negotiations to establish an International Arctic Science Committee.

The confluence of international public and political attention towards the polar regions, stemming from evolving awareness and attitudes on environmental, resource, and global change issues, poses unprecedented challenges and opportunities for the polar research community. In preparing for the decade to come, it is essential to remember that polar research figures among the most expensive research in the world and, as such, must be continuously and convincingly justified in a very competitive environment for resource allocation. It follows then, that in order to be effective and competitive, the polar research community must be highly organized, focused and capable of achieving concensus on program priorities.

The National Science Board has developed a blue print for the Foundation's future approach to polar region activities. The report, entitled "The Role of the National Science Foundation in the Polar Regions," contains recommendations which are compelling beyond NSF in a national sense and addresses more keenly our international activities. In this context, it is important to proceed, with utmost urgency, towards the development of an interagency national polar research plan, including both the Arctic and the Antarctic, as suggested by the NSB report. For the Arctic, the 5-year Arctic research plan, developed on a broad national basis under the Arctic Research and Policy Act of 1984, sets the example and standards. This needs to be followed by a similar, hopefully equally successful process, for the Antarctic. As a first step to achieve the ultimate goal of a national, bipolar research plan, the NSF Division of Polar Programs has asked the Polar Research Board of the National Academy of Sciences to take on this challenging task.

There are other, necessary developments that need to take place on a national level: we need to achieve close coordination among all interested and established actors, such as the Arctic Research Commission, the Interagency Arctic Research Policy Committee, the Interagency Arctic Policy Group, the Antarctic Policy Group, the Arctic Research Consortium, the Polar Research Board, the relevant agency advisory committees; we need to development accept new approaches, national and international, to devise the best polar research programs (special emphasis should be placed on tight management of our resources, with particular attention in instituting safety, environment and health issues); we need to transform polar activities from expeditionary to year-round programs, utilizing modern technology; we need to build bridges between the research activities dedicated to the polar regions and the emerging global programs witch will not be viable without effective polar components;

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(1)National Science Foundation, Division of Polar Programs, Washington, D.C. 20550 and, we need to devise our programs most carefully from inception, to assure that credible, scientific results will be forthcoming that are subsequently acceptable to the political, national and international decision processes.

The challenges for the next decade have been clearly identified by us, as the deciding actors regarding the research to be performed, and for us, via the political issues arising in the polar regions. We now have to forge these challenges into opportunities and articulate them cohesively to muster the political support and resources needed to carry out a task of unprecedented complexity.

A broad examination of our situation and preparedness is urgent. The decade of the 1990, which will emerge as the decade of the polar regions, is inevitable.

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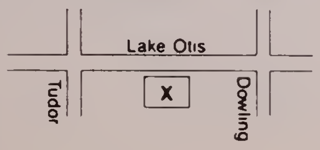

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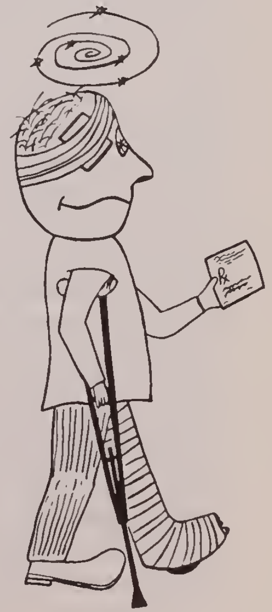

Letter to the Editor

MATERNAL AGE AND BIRTH STATISTICS

Dear Editor

Recently, a communication was sent to health care professionals from the office of the commissioner of health (1). the report outlined Alaska's relative position compared to other states with respect to infant mortality and the 'importance of prenatal care and the availability of the Alaska Healthy Baby Project'. Although the report used 1985 vital statistics, the high infant mortality rate has not improved since 1980. Teenaged mothers (<20 years) tend to have a high prevalence of low birthweigt infants and infant mortality (2). This is not surprising, since this group of mothers are likely to be unmarried, less educated and have a lower income. These variables are recognized as important contributors to high-risk pregnancy. While the need for first trimester prenatal care is apparent for all mothers, a disturbing trend of live births to unmarried mothers is emerging in Alaska.

The study population consisted of live births, maternal age and marital status at the time of birth from 1980 through 1986, compiled by the State's Section of Vital Statistics. Other variables, such as education, income, ethnic origin and birth order were not used.

This study found that there has been a progressive decline in the live birth rate trend to married mothers aged less than twenty five years (figure 1). Similar trends have been experienced by U.S. (4). In addition, figure 1 shows that the live birth rate trend for unmarried mothers has exceeded that of married mothers aged <20 years. There is an increasing trend of live births to unmarried mothers in general (figure 2). Much interest has been expressed in improving Alaska's infant mortality rate. While neonatal mortality is accepted as an indicator of the adequacy of prenatal care and postneonatal mortality as an indicator of infant health, both of these rates are directly related to maternal age and marital status (2). Since 1980 there has been a progressive increase (from 9% to 21%) in the proportion of live births to unmarried mothers in Alaska. This pattern has been recognized for the U.S. as a whole since 1981 (3,5). In 1985, for example, U.S. experienced numerical live birth rate of 22 percent (range, 15-25 per 1000 live births). In Alaska, 58% of live births to women aged <20 years are born to unmarried mothers. Approximately 50% of mothers aged <20 will conceive a second time within two years after the termination of their first pregnancy. The rising birth rate to unmarried mothers may exact its toll by placing greater burden on social welfare programs, tertiary neonatal services and slowing improvement in infant mortality rate. The trend of increasing birth rate to unmarried young mothers is disturbing to health care professionals who must interact with children having babies. While providing prenatal care service to an ever expanding group of young mothers is laudable, perhaps priorities for teenagers should focus on pre-pregnancy planning, school retention and higher state expenditures for public welfare programs. These factors have been positively associated with decline in teenage pregnancy (6).

Harry Harrison, Jr., M.D.
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Anchorage, Alaska 99508

Figures 1 and 2 on following page.

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Figure 1

Live births by marital status and age, 1980-86

■——■ Unmarried mothers

○——○ Married mothers

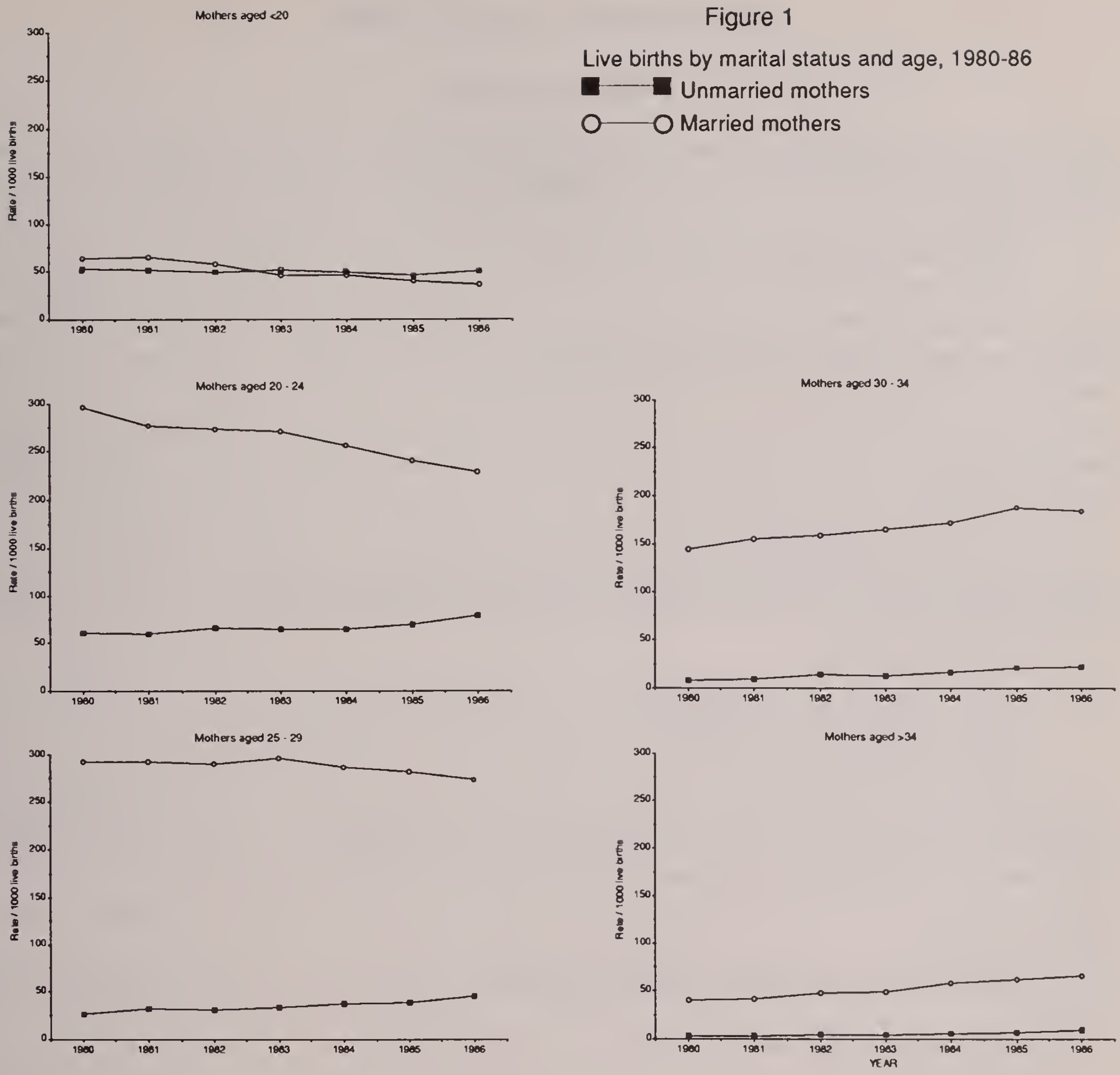
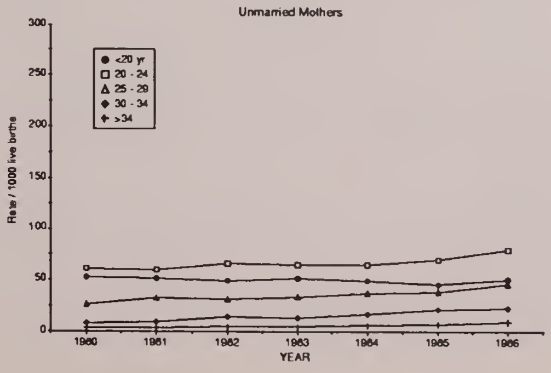


Figure 2

Live births for Unmarried mothers by age, 1980-86



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Warnings: *Angioedema:* Angioedema of the face, extremities, lips, tongue, glottis, and/or larynx has been reported in patients treated with ACE inhibitors, including VASOTEC. In such cases, VASOTEC should be promptly discontinued and the patient carefully observed until the swelling disappears. In instances where swelling has been confined to the face and lips, the condition has generally resolved without treatment, although antihistamines have been useful in relieving symptoms. Angioedema associated with laryngeal edema may be fatal. **Where there is involvement of the tongue, glottis, or larynx likely to cause airway obstruction, appropriate therapy, e.g., subcutaneous epinephrine solution 1:1000 (0.3 mL to 0.5 mL), should be promptly administered.** (See ADVERSE REACTIONS.)

Hypotension: Excessive hypotension is rare in uncomplicated hypertensive patients treated with VASOTEC alone. Patients with heart failure given VASOTEC commonly have some reduction in blood pressure, especially with the first dose, but discontinuation of therapy for continuing symptomatic hypotension usually is not necessary when dosing instructions are followed; caution should be observed when initiating therapy. (See DOSAGE AND ADMINISTRATION.) Patients at risk for excessive hypotension, sometimes associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death, include those with the following conditions or characteristics: heart failure, hyponatremia, high-dose diuretic therapy, recent intensive diuresis or increase in diuretic dose, renal dialysis, or severe volume and/or salt depletion of any etiology. It may be advisable to eliminate the diuretic (except in patients with heart failure), reduce the diuretic dose, or increase salt intake cautiously before initiating therapy with VASOTEC in patients at risk for excessive hypotension who are able to tolerate such adjustments. (See PRECAUTIONS, *Drug Interactions* and ADVERSE REACTIONS.) In patients at risk for excessive hypotension, therapy should be started under very close medical supervision and such patients should be followed closely for the first two weeks of treatment and whenever the dose of enalapril and/or diuretic is increased. Similar considerations may apply to patients with ischemic heart disease or cardiovascular disease in whom an excessive fall in blood pressure could result in a myocardial infarction or cerebrovascular accident. If excessive hypotension occurs, the patient should be placed in the supine position and, if necessary, receive an intravenous infusion of normal saline. A transient hypotensive response is not a contraindication to further doses of VASOTEC, which usually can be given without difficulty once the blood pressure has stabilized. If symptomatic hypotension develops, a dose reduction or discontinuation of VASOTEC or concomitant diuretic may be necessary.

Neutropenia/Agranulocytosis: Another ACE inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression, rarely in uncomplicated patients but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease. Available data from clinical trials of enalapril are insufficient to show that enalapril does not cause agranulocytosis at similar rates. Foreign marketing experience has revealed several cases of neutropenia or agranulocytosis in which a causal relationship to enalapril cannot be excluded. Periodic monitoring of white blood cell counts in patients with collagen vascular disease and renal disease should be considered.

Precautions: *General: Impaired Renal Function:* As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including VASOTEC, may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death.

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine were observed in 20% of patients. These increases were almost always reversible upon discontinuation of enalapril and/or diuretic therapy. In such patients, renal function should be monitored during the first few weeks of therapy.

Some patients with hypertension or heart failure with no apparent preexisting renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when VASOTEC has been given concomitantly with a diuretic. This is more likely to occur in patients with preexisting renal impairment. Dosage reduction and/or discontinuation of the diuretic and/or VASOTEC may be required.

Evaluation of patients with hypertension or heart failure should always include assessment of renal function. (See DOSAGE AND ADMINISTRATION.)

Hyperkalemia: Elevated serum potassium (>5.7 mEq/L) was observed in approximately 1% of hypertensive patients in clinical trials. In most cases these were isolated values which resolved despite continued therapy. Hyperkalemia was a cause of discontinuation of therapy in 0.28% of hypertensive patients. In clinical trials in heart failure, hyperkalemia was observed in 3.8% of patients, but was not a cause for discontinuation.

Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with VASOTEC. (See *Drug Interactions*.)

Surgery/Anesthesia: In patients undergoing major surgery or during anesthesia with agents that produce hypotension, enalapril may block angiotensin II formation secondary to compensatory renin release. If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion.

Information for Patients.

Angioedema: Angioedema, including laryngeal edema, may occur especially following the first dose of enalapril. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to take no more drug until they have consulted with the prescribing physician.

Hypotension. Patients should be cautioned to report lightheadedness, especially during the first few days of therapy. If actual syncope occurs, the patients should be told to discontinue the drug until they have consulted with the prescribing physician.

All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduction in fluid volume. Other causes of volume depletion such as vomiting or diarrhea may also lead to a fall in blood pressure; patients should be advised to consult with the physician.

Hyperkalemia: Patients should be told not to use salt substitutes containing potassium without consulting their physician.

Neutropenia: Patients should be told to report promptly any indication of infection (e.g., sore throat, fever) which may be a sign of neutropenia.

NOTE: As with many other drugs, certain advice to patients being treated with enalapril is warranted. This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects.

Drug Interactions

Hypotension. Patients on Diuretic Therapy: Patients on diuretics and especially those in whom diuretic therapy was recently instituted may occasionally experience an excessive reduction of blood pressure after initiation of therapy with enalapril. The possibility of hypotensive effects with enalapril can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with enalapril. If it is necessary to continue the diuretic, provide close medical supervision after the initial dose for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and DOSAGE AND ADMINISTRATION.)

Agents Causing Renin Release: The antihypertensive effect of VASOTEC is augmented by antihypertensive agents that cause renin release (e.g., diuretics).

Other Cardiovascular Agents. VASOTEC has been used concomitantly with beta-adrenergic-blocking agents, methyldopa, nitrates, calcium-blocking agents, hydralazine, prazosin, and digoxin without evidence of clinically significant adverse interactions.

Agents Increasing Serum Potassium: VASOTEC attenuates potassium loss caused by thiazide-type diuretics. Potassium-sparing diuretics (e.g., spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium. Therefore, if concomitant use of these agents is indicated because of demonstrated hypokalemia, they should be used with caution and with frequent monitoring of serum potassium. Potassium-sparing agents should generally not be used in patients with heart failure receiving VASOTEC.

Lithium: Lithium toxicity has been reported in patients receiving lithium concomitantly with drugs which cause elimination of sodium, including ACE inhibitors. A few cases of lithium toxicity have been reported in patients receiving concomitant VASOTEC and lithium and were reversible upon discontinuation of both drugs. It is recommended that serum lithium levels be monitored frequently if enalapril is administered concomitantly with lithium.

Pregnancy—Category C. There was no fetotoxicity or teratogenicity in rats treated with up to 200 mg/kg/day of enalapril (333 times the maximum human dose). Fetotoxicity, expressed as a decrease in average fetal weight, occurred in rats given 1200 mg/kg/day of enalapril but did not occur when these animals were supplemented with saline. Enalapril was not teratogenic in rabbits. However, maternal and fetal toxicity occurred in some rabbits at doses of 1 mg/kg/day or more. Saline supplementation prevented the maternal and fetal toxicity seen at doses of 3 and 10 mg/kg/day, but not at 30 mg/kg/day (50 times the maximum human dose).

Radioactivity was found to cross the placenta following administration of labeled enalapril to pregnant hamsters.

There are no adequate and well-controlled studies of enalapril in pregnant women. However, data are available that show enalapril crosses the human placenta. Because the risk of fetal toxicity with the use of ACE inhibitors has not been clearly defined, VASOTEC® (Enalapril Maleate, MSD) should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Postmarketing experience with all ACE inhibitors thus far suggests the following with regard to pregnancy outcome. Inadvertent exposure limited to the first trimester of pregnancy has not been reported to affect fetal outcome adversely. Fetal exposure during the second and third trimesters of pregnancy has been associated with fetal and neonatal morbidity and mortality.

When ACE inhibitors are used during the later stages of pregnancy, there have been reports of hypotension and decreased renal perfusion in the newborn. Oligohydramnios in the mother has also been reported, presumably representing decreased renal function in the fetus. Infants exposed *in utero* to ACE inhibitors should be closely observed for hypotension, oliguria, and hyperkalemia. If oliguria occurs, attention should be directed toward support of blood pressure and renal perfusion with the administration of fluids and pressors as appropriate. Problems associated with prematurity such as patent ductus arteriosus have occurred in association with maternal use of ACE inhibitors, but it is not clear whether they are related to ACE inhibition, maternal hypertension, or the underlying prematurity.

Nursing Mothers: Milk in lactating rats contains radioactivity following administration of ^{14}C enalapril maleate. It is not known whether this drug is secreted in human milk. Because many drugs are secreted in human milk, caution should be exercised when VASOTEC is given to a nursing mother.

Pediatric Use: Safety and effectiveness in children have not been established.

Adverse Reactions: VASOTEC has been evaluated for safety in more than 10,000 patients, including over 1000 patients treated for one year or more. VASOTEC has been found to be generally well tolerated in controlled clinical trials involving 2987 patients.

HYPERTENSION: The most frequent clinical adverse experiences in controlled trials were: headache (5.2%), dizziness (4.3%), and fatigue (3%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in controlled clinical trials were: diarrhea (1.4%), nausea (1.4%), rash (1.4%), cough (1.3%), orthostatic effects (1.2%), and asthenia (1.1%).

HEART FAILURE: The most frequent clinical adverse experiences in both controlled and uncontrolled trials were: dizziness (7.9%), hypotension (6.7%), orthostatic effects (2.2%), syncope (2.2%), cough (2.2%), chest pain (2.1%), and diarrhea (2.1%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in both controlled and uncontrolled clinical trials were: fatigue (1.8%), headache (1.8%), abdominal pain (1.6%), asthenia (1.6%), orthostatic hypotension (1.6%), vertigo (1.6%), angina pectoris (1.5%), nausea (1.3%), vomiting (1.3%), bronchitis (1.3%), dyspnea (1.3%), urinary tract infection (1.3%), rash (1.3%), and myocardial infarction (1.2%).

Other serious clinical adverse experiences occurring since the drug was marketed or adverse experiences occurring in 0.5% to 1% of patients with hypertension or heart failure in clinical trials in order of decreasing severity within each category:

Cardiovascular. Cardiac arrest; myocardial infarction or cerebrovascular accident, possibly secondary to excessive hypotension in high-risk patients (see WARNINGS, *Hypotension*); cardiac arrest; pulmonary embolism and infarction; rhythm disturbances; atrial fibrillation; palpitation.

Digestive: Ileus, pancreatitis, hepatitis or cholestatic jaundice, melena, anorexia, dyspepsia, constipation, glossitis, stomatitis.

Musculoskeletal: Muscle cramps.

Nervous/Psychiatric: Depression, confusion, ataxia, somnolence, insomnia, nervousness, paresthesia.

Urogenital: Renal failure, oliguria, renal dysfunction (see PRECAUTIONS and DOSAGE AND ADMINISTRATION).

Respiratory: Bronchospasm, rhinorrhea, sore throat and hoarseness, asthma, upper respiratory infection.

Skin: Herpes zoster, urticaria, pruritus, alopecia, flushing, hyperhidrosis.

Special Senses: Blurred vision, taste alteration, tinnitus.

A symptom complex has been reported which may include a positive ANA, an elevated erythrocyte sedimentation rate, arthralgias/arthritis, myalgias, fever, serositis, vasculitis, leukocytosis, eosinophilia, photosensitivity, rash and other dermatologic manifestations.

Angioedema: Angioedema has been reported in patients receiving VASOTEC (0.2%). Angioedema associated with laryngeal edema may be fatal. If angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with VASOTEC should be discontinued and appropriate therapy instituted immediately. (See WARNINGS.)

Hypotension: In the hypertensive patients, hypotension occurred in 0.9% and syncope occurred in 0.5% of patients following the initial dose or during extended therapy. Hypotension or syncope was a cause for discontinuation of therapy in 0.1% of hypertensive patients. In heart failure patients, hypotension occurred in 6.7% and syncope occurred in 2.2% of patients. Hypotension or syncope was a cause for discontinuation of therapy in 1.9% of patients with heart failure. (See WARNINGS.)

Clinical Laboratory Test Findings

Serum Electrolytes: Hyperkalemia (see PRECAUTIONS), hyponatremia.

Creatinine, Blood Urea Nitrogen: In controlled clinical trials, minor increases in blood urea nitrogen and serum creatinine, reversible upon discontinuation of therapy, were observed in about 0.2% of patients with essential hypertension treated with VASOTEC alone. Increases are more likely to occur in patients receiving concomitant diuretics or in patients with renal artery stenosis. (See PRECAUTIONS.) In patients with heart failure who were also receiving diuretics with or without digitalis, increases in blood urea nitrogen or serum creatinine, usually reversible upon discontinuation of VASOTEC and/or other concomitant diuretic therapy, were observed in about 11% of patients. Increases in blood urea nitrogen or creatinine were a cause for discontinuation in 1.2% of patients.

Hemoglobin and Hematocrit: Small decreases in hemoglobin and hematocrit (mean decreases of approximately 0.3 g% and 1.0 vol %, respectively) occur frequently in either hypertension or heart failure patients treated with VASOTEC but are rarely of clinical importance unless another cause of anemia coexists. In clinical trials, less than 0.1% of patients discontinued therapy due to anemia.

Other (Causal Relationship Unknown): In marketing experience, rare cases of neutropenia, thr[illegible]a, and bone marrow depression have been reported. A few cases of hemolysis have been reported in pati[illegible] G6PD deficiency.

Liver Function Tests: Elevations of liver enzymes and/or serum bilirubin have occurred.

Dosage and Administration: *Hypertension:* In patients who are currently be[illegible] treated with a diuretic, symptomatic hypotension occasionally may occur following the initial dose of VASOTEC. [illegible] diuretic should, if possible, be discontinued for two to three days before beginning therapy with VASOTEC to reduce the [illegible]lihood of hypotension. [illegible] WARNINGS.) If the patient's blood pressure is not controlled with VASOTEC alone, diur[illegible]erapy may be res[illegible].

If the diuretic cannot be discontinued, an initial dose of 2.5 mg should be used under medical s[illegible]vision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions*.)

The recommended initial dose in patients not on diuretics is 5 mg once a day. Dosage should be adjusted acco[illegible] to blood pressure response. The usual dosage range is 10 to 40 mg per day administered in a single dose or in two divided doses. In some patients treated once daily, the antihypertensive effect may diminish toward the end of the dosing interval. In such patients, an increase in dosage or twice-daily administration should be considered. If blood pressure is not controlled with VASOTEC alone, a diuretic may be added.

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Concomitant administration of VASOTEC with potassium supplements, potassium salt substitutes, or potassium-sparing diuretics may lead to increases of serum potassium (see PRECAUTIONS).

Dosage Adjustment in Hypertensive Patients with Renal Impairment: The usual dose of enalapril is recommended for patients with a creatinine clearance > 30 mL/min (serum creatinine of up to approximately 3 mg/dL). For patients with creatinine clearance ≤ 30 mL/min (serum creatinine ≥ 3 mg/dL), the first dose is 2.5 mg once daily. The dosage may be titrated upward until blood pressure is controlled or to a maximum of 40 mg daily.

Heart Failure: VASOTEC is indicated as adjunctive therapy with diuretics and digitalis. The recommended starting dose is 2.5 mg once or twice daily. After the initial dose of VASOTEC, the patient should be observed under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions*.) If possible, the dose of the diuretic should be reduced, which may diminish the likelihood of hypotension. The appearance of hypotension after the initial dose of VASOTEC does not preclude subsequent careful dose titration with the drug, following effective management of the hypotension. The usual therapeutic dosing range for the treatment of heart failure is 5 to 20 mg daily given in two divided doses. The maximum daily dose is 40 mg. Once-daily dosing has been effective in a controlled study, but nearly all patients in this study were given 40 mg, the maximum recommended daily dose, and there has been much more experience with twice-daily dosing. In addition, in a placebo-controlled study which demonstrated reduced mortality in patients with severe heart failure (NYHA Class IV), patients were treated with 2.5 to 40 mg per day of VASOTEC, almost always administered in two divided doses. (See CLINICAL PHARMACOLOGY, *Pharmacodynamics and Clinical Effects*.) Dosage may be adjusted depending upon clinical or hemodynamic response. (See WARNINGS.)

Dosage Adjustment in Patients with Heart Failure and Renal Impairment or Hyponatremia: In patients with heart failure who have hyponatremia (serum sodium < 130 mEq/L) or with serum creatinine >1.6 mg/dL, therapy should be initiated at 2.5 mg daily under close medical supervision. (See DOSAGE AND ADMINISTRATION, *Heart Failure*, WARNINGS, and PRECAUTIONS, *Drug Interactions*.) The dose may be increased to 2.5 mg b.i.d., then 5 mg b.i.d. and higher as needed, usually at intervals of four days or more, if at the time of dosage adjustment there is not excessive hypotension or significant deterioration of renal function. The maximum daily dose is 40 mg.

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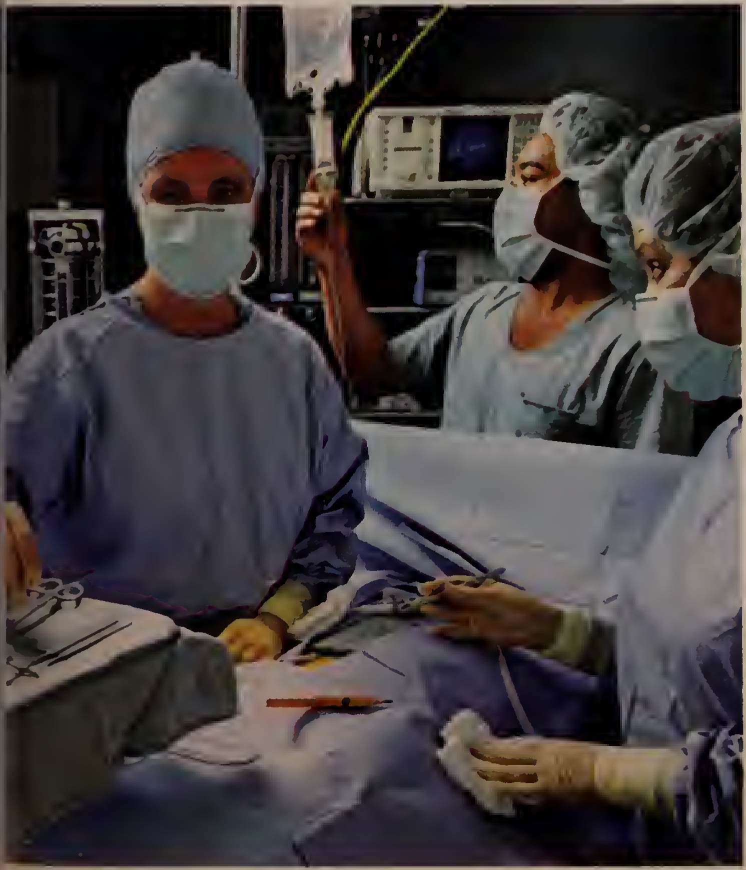

Dr. Holwick in operating room at Letterman Army Medical Center.

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General and Trauma Surgeon.
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HOSPITAL AFFILIATIONS St. Luke Hospital; Huntington Memorial Hospital, Pasadena, California; Traumatologist, Arcadia Methodist Hospital, Arcadia, California.

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5. Am J Gastroenterol 1989;84:769-774.

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Precautions: *General*—1. Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy.

2. Dosage should be reduced in patients with moderate to severe renal insufficiency.

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Carcinogenesis, Mutagenesis, Impairment of Fertility—A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups. The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

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Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a two-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy—Teratogenic Effects—Pregnancy Category C—Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in one fetus, and at 50 mg/kg, it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in one fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only it the potential benefit justifies the potential risk to the fetus.

Nursing Mothers—Studies in lactating women have shown that 0.1% of an oral dose is secreted in human milk in proportion to plasma concentrations. Because of growth depression in pups reared by treated lactating rats, a decision should be made whether to discontinue nursing or the drug, taking into account the importance of the drug to the mother.

Pediatric Use—Safety and effectiveness in children have not been established.

Use in Elderly Patients—Healing rates in elderly patients were similar to those in younger age groups as were the rates of adverse events and laboratory test abnormalities. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Clinical trials of varying durations included almost 5,000 patients. Among the more common adverse events in domestic placebo-controlled trials of over 1,900 nizatidine patients and over 1,300 on placebo, sweating (1% vs 0.2%), urticaria (0.5% vs <0.01%), and somnolence (2.4% vs 1.3%) were significantly more common with nizatidine. It was not possible to determine whether a variety of less common events was due to the drug.

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Hepatic—Hepatocellular injury (elevated liver enzyme tests or alkaline phosphatase) possibly or probably related to nizatidine occurred in some patients. In some cases, there was marked elevation (>500 IU/L) in SGOT or SGPT and, in a single instance, SGPT was >2,000 IU/L. The incidence of elevated liver enzymes overall and elevations of up to three times the upper limit of normal, however, did not significantly differ from that in placebo patients. Hepatitis and jaundice have been reported. All abnormalities were reversible after discontinuation of Axid.

Cardiovascular—In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in two individuals administered Axid and in three untreated subjects.

CNS—Rare cases of reversible mental confusion have been reported.

Endocrine—Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to nizatidine. Impotence and decreased libido were reported with equal frequency by patients on nizatidine and those on placebo. Gynecomastia has been reported rarely.

Hematologic—Fatal thrombocytopenia was reported in a patient treated with nizatidine and another H_2-receptor antagonist. This patient had previously experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental—Sweating and urticaria were reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity—As with other H_2-receptor antagonists, rare cases of anaphylaxis following nizatidine administration have been reported. Because cross-sensitivity among this class has been observed, H_2-receptor antagonists should not be administered to those with a history of hypersensitivity to these agents. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other—Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine have been reported.

Overdosage: Overdoses of Axid have been reported rarely. If overdosage occurs, activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. Renal dialysis for four to six hours increased plasma clearance by approximately 84%.

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Young girl sitting in front of her home (insert) in Siberian village at Lake Baikal near Irkutsk. Photo courtesy of Theodore Mala, M.D., M.P.H., Director, Institute for Circumpolar Health Studies, University of Alaska Anchorage.

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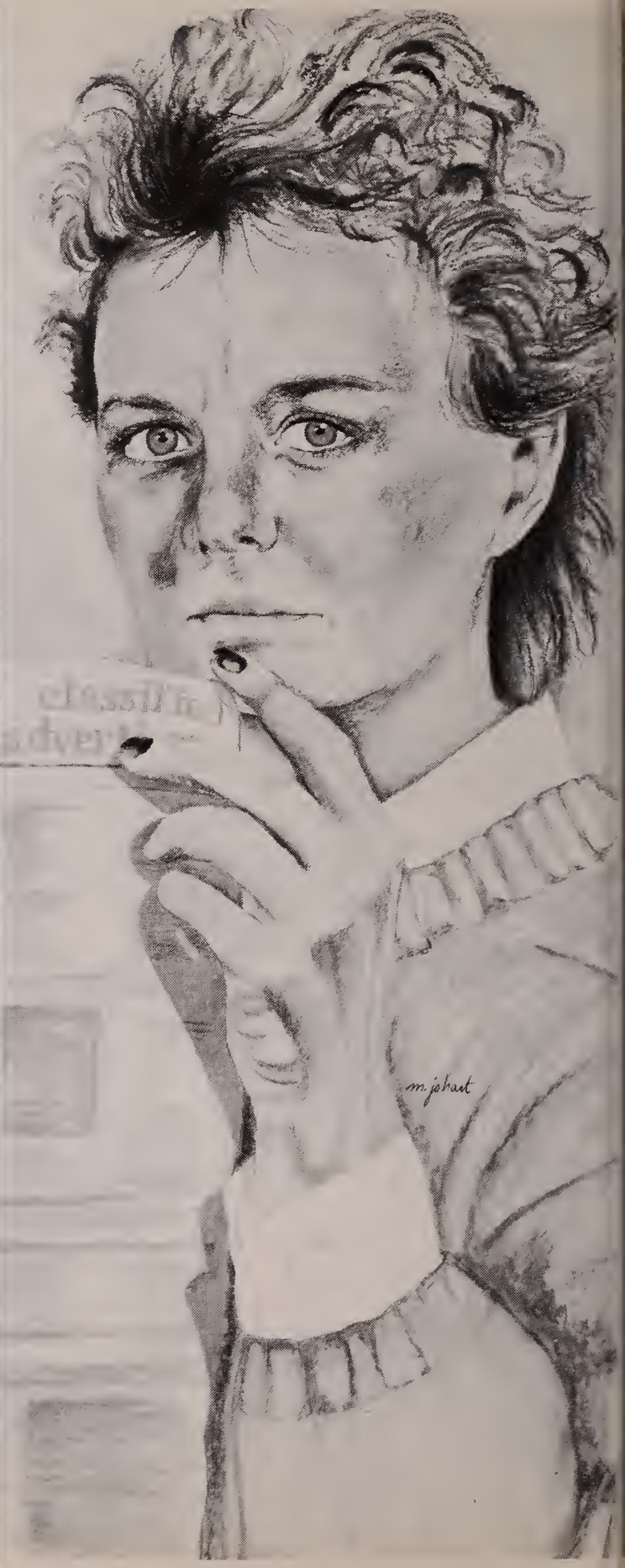

Death in Anchorage in 1987

by Rodman Wilson, M.D.(1)

ABSTRACT

In 1987, 855 persons died in Anchorage, Alaska. Crude death rate was slightly higher than in 1986. Age-adjusted death rate was distinctly higher than in the nation.

Proportions of deaths due to cardiovascular disease, cancer, violence, and other categories were about the same as in 1986.

INTRODUCTION

Eight-hundred fifty-five persons died within the boundaries of the Municipality of Anchorage, Alaska, in 1987. This was 32 fewer than died in 1986,* when 17,022 more people were estimated to be living in the city(1).

METHOD

Information was collected from death certificates and interviews with physicians in the same manner as in previous studies of deaths in 1985 and 1986(2,3). Autopsy had been done in 271 (31.6%) cases, most commonly after violent death. Physicians were spoken to in 209 (24.4%) cases to clarify the underlying cause of death. The value of such query has recently been demonstrated in Oregon(4).

RESULTS

Deaths by underlying cause are listed in the Appendix.

Median age at death was 60 years, the same as in 1986. Mean age at death, not including deaths before age 1, however, rose from 58.0 years in 1986 to 58.5 years in 1987.

Crude death rate in 1987 was 3.73 deaths per 1,000 persons in Anchorage. In the nation the crude death rate for 1987 was 8.72(5). When both populations are "age-adjusted" to correct for differing distributions by age groups (using the "direct" method and the 1940 U.S. Census as the standard population), the Anchorage age-adjusted death rate in 1987 was 6.75 and the rate in the nation only 5.4(5).

Both crude and age-adjusted death rates for Anchorage must be interpreted cautiously, for many sick and injured persons are brought to Anchorage and die here while far fewer Anchorage residents die away from the city.+ The population base within which persons die in Anchorage is thus uncertain. Rates, therefore, are imprecise(2,3).

Proportions of deaths by broad categories of cause such as cardiovascular disease (31.0%), cancer (24.8%), violence as previously defined (18.5%), death before age 1 (7.4%), lung disease (6.3%), and infection (4.1%) were little changed from 1986(3).

Coronary heart disease was the preeminent killer in 1987, causing 153 (17.8%) deaths. Deaths from carcinoma of the lung were higher than ever - 71 (8.3%), 33 of them among women. Alaska leads the nation in rate of death from this malignancy(6).

Nineteen persons died from motor vehicle impacts in Anchorage in 1987 compared to 27 in 1986 and 50** in 1985. Eight of the 19 were pedestrians, the same number as in 1986. Six other auto victims, including another pedestrian, died in Anchorage hospitals after collisions elsewhere.

Thirty (3.5%) persons died from gunfire in 1987, 16 suicidally, 10 homicidally, 3 unintentionally, and 1 inexplicably.

In all, there were 30 suicides and 19 homicides in 1987. Five persons died directly from use of cocaine (3), heroin (1), or morphine (1).

There were fewer deaths due to immaturity at birth (21) in 1987 than in 1986 (31).

COMMENT

Proportions of death by broad categories such as cardiovascular disease, cancer, violence, death before age 1, lung disease, and infection were approximately the same as in 1986.

---

(1)6234 Tanaina Drive, Anchorage, Alaska 99502.

* Since reporting 887 deaths in Anchorage in 1986(3) another death for that year has been recorded: an 18-year-old Caucasian male soldier from Ohio walked onto Knik Arm ice and apparently fell into a sink hole.

+ In 1987, 220 non-residents died in Anchorage; 71 Anchorage residents are known to have succumbed elsewhere in Alaska or out-of-state, and more probably did.

Violent deaths, however, fell sharply from 25.5% of all deaths in 1985 to 18.5% in 1987 ($p < 0.005$). Even at this lower proportion, violent demise remains approximately three times as common in Anchorage, on an unadjusted basis, as in the nation.

Whether the decline in numbers of violent deaths in Anchorage between 1985 and 1987 represents a trend will be further scrutinized when 1988 deaths are collated.

ACKNOWLEDGMENTS

This study was done with the permission and assistance of the Municipality of Anchorage Department of Health & Human Services, Bert Hall, Director. Rose Courtright prepared the Appendix.

** Including one death in Anchorage recently discovered to have been incorrectly recorded in Palmer, AK, in 1985.

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APPENDIX

Underlying Cause of Death of 855 Persons

Dying in Anchorage in 1987

(code numbers are from ICD-9-CM, 3rd ed., 1989)

CARDIOVASCULAR DISEASE				265 (31.0%)
Heart Disease*			176	
coronary artery(410,414)		153		
acute myocardial infarction (410)	73			
rheumatic(393-398)		6		
cardiomyopathy, idiopathic(425.4)		6		
aortic valvular stenosis, idiopathic(424.1)		4		
hypertensive(402)		2		
primary pulmonary hypertensive(416.0)		2		
cardiac arrest, unexplained(427.4,427.5)		2		
trifascicular block, cause undet(426.54)		1		
*excluding alcoholic cardiomyopathy, infections of the heart, and death from heart disease before age 1				
Vascular Disease			89	
cerebral thrombosis(434.0)		35		
cerebral hemorrhage		19		
intraparenchymal(431)	10			
aneurysm(430)	9			
aortic aneurysm, abdominal(441.3,441.4)		10		
atherosclerosis, generalized(440.9)		10		

pulmonary embolism(415.1)	10		
vascular insufficiency of intestine(557)	4		
atherosclerosis of vessels of extremities(440.2)	1		
CANCER			212 (24.8%)
Lung(162)		71	
Colo-rectal(153,154)		18	
Breast(174)		11	
Pancreas(157)		10	
Myeloid leukemia(205)		9	
Brain(191)		8	
Disseminated, primary site unspecified(199.0)		8	
Stomach(151)		7	
Ovary(183)		7	
Prostate(185)		7	
Melanoma(172)		7	
Lymphoma(200-202)		7	
Oral and nasopharyngeal(146-149)		5	
Tongue(141)		4	
Bladder(188)		3	
Cervix(180)		3	
Myeloma(203)		3	
Myeloproliferative disorder(238.7)		2	
Larynx(161)		2	
Esophagus(150)		2	
Esophago-gastric junction(151.0)		2	
Bile ducts, extrahepatic(156.1)		2	
Kidney(189)		2	
Bone(170)		2	
Lymphoid leukemia(204)		1	
Hairy cell leukemia(202.4)		1	
Duodenum(152.0)		1	
Liver, primary(155.0)		1	
Penis(187)		1	
Endometrial(182)		1	
Choriocarcinoma(181)		1	
Spinal cord(192.2)		1	
Connective tissue, origin undet(171.8)		1	
Liposarcoma, thigh(171.3)		1	
VIOLENCE AND ADVERSE EFFECTS			158 (18.5%)
Alcoholism*		40	
alcoholic liver disease(571.0-157.3)	22		
acute alcoholism(303.0)	6		
alcoholic cardiomyopathy(425.5)	5		
hypothermia, drunk(991.6)	2		
fall down stairs, drunk(E880)	2		
aspiration, drunk(507.0)	1		
acute pancreatis, alcoholic(577.0)	1		
chronic alcoholism(303.9)	1		
*not including fetal alcohol syndrome			
Vehicular Accidents		32	
automobile(E811,812,815,816)	14		

auto-pedestrian(E814)	9		
fall from truck, crushed(E818)	1		
motorcycle(E812.2)	1		
snowmachine(E820)	1		
bicycle(E826)	1		
aircraft(E841)	5		
Suicide		30	
gunshot(E955)	16		
hanging(E953.0)	6		
motor vehicle exhaust(E952.0)	4		
jumping(E957)	1		
fire(E958.1)	1		
drug, morphine(E950.0)	1		
chemical, ingestion of caustic(E958.7)	1		
Homicide		19	
gunshot(E965)	10		
strangulation(E963)	5		
stabbing or cutting(E966)	3		
striking(E968.2)	1		
Drowning(E910)		7	
Poisoning, unintentional		6	
cocaine(E855.2)	3		
heroin(E850.0)	1		
morphine(E850.2)	1		
methanol(E860.2)	1		
Fire(E890)		5	
Fall*		5	
on job(E888)	2		
from bed(E884.2)	2		
hiking(E886.0)	1		
*not including fall while drunk			
Crushing or cave-in, not on job(E913,E916)		4	
Blow, accidental(E917)		3	
Gunshot, unintentional(E922)		3	
Electrocution(E925)		2	
on job	1		
hang-gliding	1		
Gunshot, undetermined whether suicide, homicide or unintentional (E985)		1	
Hanging, undetermined whether suicide or homicide(E983.0)		1	
DEATH BEFORE AGE 1*			63 (7.4%)
Immaturity at birth(765)		21	
Sudden infant death syndrome(798.0)		16	
Congenital anomalies(740-759)		8	
trisomy 18(758.2)	1		
lethal chromosomal abnormality(758.9)	1		
diaphragmatic hernia(756.6)	1		
osteogenesis imperfecta(756.51)	1		
congenital hydrocephalus(742.3)	1		
tetrology of Fallot(745.2)	1		
multiple anomalies of heart(746.9)	1		
multiple congenital anomalies(759.7)	1		

Birth asphyxia(768)		4	
Respiratory distress syndrome(769,770.8)		2	
Meningitis		2	
salmonella(003.21)	1		
streptococcal(320.2)	1		
Pneumonia(486)		1	
Necrotizing enterocolitis(777.5)		1	
Prolapse of umbilical cord(762.4)		1	
Hydrops fetalis(778.0)		1	
No neurological activity(779.0)		1	
Fetal alcohol syndrome(760.71)		1	
Asphyxia, undetermined whether unintentional or homicide(E983)		1	
Congenital cardiac arrhythmia(746.9)		1	
Infantile spinal muscular atrophy(335.0)		1	
Unknown cause(799.9)		1	

*not including 1 death from drowning

LUNG DISEASE*			54 (6.3%)
Chronic obstructive pulmonary disease(496)+		43	
Asthma(493)		4	
Idiopathic fibrosing alveolitis(516.3)		4	
Bronchiectasis(494)		1	
Restrictive lung disease secondary to tuberculosis(011.4)		1	
Restrictive lung disease secondary to kyphoscoliosis(737.3)		1	

*excluding pneumonia, lung abscess, and pulmonary disorders of birth immaturity

+COPD was listed on 41 other certificates as a significant associated condition

INFECTION*			35 (4.1%)
Pneumonia(486)		11	
Acquired immunodeficiency syndrome (042)		7	
Septicemia(038)		5	
Pyelonephritis(590.80)		3	
Viral hepatitis, non A, non B(070.5)		3	
Pneumococcal meningitis, late effects(320.1)		1	
Serratia meningitis, late effects(320.8)		1	
Chicken pox(052)		1	
Lung abscess(513)		1	
Viral myocarditis(422)		1	
Subacute bacterial endocarditis(421.0)		1	

*not including death from infection before age 1

NEUROLOGIC DISEASE*			32 (3.7%)
Senile dementia and Alzheimer's disease(290.0,330.1)		9	
Multi-infarct arteriosclerotic dementia(290.4)		7	
Parkinson disease(332)		3	
Epilepsy(345)		3	
Amyotrophic lateral sclerosis(335.20)		2	
Multiple sclerosis(340)		2	
Demyelinating disease of CNS, unspecified(341.9)		1	
Syringomyelia(336.0)		1	

Congenital hydrocephalus(742.3)	1	
Infantile quadriplegia(343.2)	1	
Psychosis, organic, not classified(294)	1	
Convulsion, unexplained(780.3)	1	

*excluding cerebral thrombosis, cerebral hemorrhage, brain tumor, and deaths from neurologic disease before age 1

GASTROINTESTINAL		16 (1.9%)
Acute pancreatitis, non-alcoholic(577.0)	2	
Cholangitis(576.1)	2	
Appendicitis, with abscess(540.1)	2	
Diverticulitis of colon, perforated (562.11)	2	
Perforation of colon, cause undetermined(569.83)	1	
Esophagitis(530.1)	1	
Duodenal ulcer, perforated(532.5)	1	
Choletithiasis(574)	1	
Umbilical hernia, incarcerated(552.1)	1	
Inguinal hernia, incarcerated(550.1)	1	
Intestinal obstruction, due to adhesions(560.81)	1	
Inanition, due to short bowel syndrome(579.3)	1	
MISCELLANEOUS		20 (2.3%)
Senility(797)	3	
Diabetic end-stage renal disease(250.4)*	3	
Chronic glomerulonephritis, end stage renal disease(582)	3	
Interstitial nephritis, end-stage renal disease(583.89)	1	
Gangrene of foot, secondary to diabetes mellitus(250.7)	1	
Rheumatoid arthritis, (714.0)	1	
Systemic lupus erythematosus(710.0)	1	
Systemic sclerosis(710.1)	1	
Tuberous sclerosis(759.5)	1	
Cystic fibrosis(277.0)	1	
Perfusion of heart with water unintentional(E870.1)	1	
Drug toxicity, isoniazide(E931.8)	1	
Unknown cause(799.9)	2	

*Diabetes mellitus was mentioned a total of 53 times (6.1%) among 855 deaths

	Total	855 (100.0%)

The Federal Government - Native American Relationship and the Indian Health Service in Alaska

by Penelope M. Cordes, Ph.D(1)

INTRODUCTION

The United States government provides health care to Native Americans through the Indian Health Service (IHS), a division of the Public Health Service, Department of Health and Human Services. Alaska Natives are eligible to receive health care through IHS if they are affiliated with a tribal group, Alaska Native village or regional corporation, or they are descended from someone so affiliated. Presently there are approximately 90,000 Alaska Natives who are beneficiaries of the Alaska Area Native Health Services. Non-Natives are eligible for services under certain circumstances. A non-Native woman pregnant with a child of an IHS beneficiary is eligible for pregnancy, delivery and postpartum health care. A non-Native child adopted by an IHS beneficiary is eligible for services until age nineteen. Non-Native household members of beneficiaries may be temporarily eligible for services for purposes of communicable disease control(1). The provision of health care and other social service programs exclusively for the benefit of American Indians and Alaska Natives is based on the unique legal relationship between the federal government and Native Americans.

THE UNIQUE LEGAL RELATIONSHIP

The legal relationship between the federal government and American Indians and Alaska Natives has developed from: treaties which have established "trust responsibilities"; court decisions which have defined the nature of the political relationship between Native Americans and the U.S. government; and a history of statutes, appropriations, executive actions, and court decisions which have acknowledged the legal obligations of the government toward Native Americans.

The concept of trust responsibility stems from treaties negotiated prior to 1871 whereby Native American tribes ceded tracts of lands to the U.S. government in return for promised goods and services; or agreements whereby the government holds Indian lands in trust, providing services with funds obtained from the sale or lease of the resources of those lands.

Early Supreme Court decisions by Chief Justice John Marshall confirmed the assumptions upon which treaties were made: that Indian tribes are sovereign entities, and that, however unequal, the relationship between tribes and the United States was that of government to government. In Worchester v. Georgia (1830) the court ruled that Indian tribes were "distinct, independent political communities having territorial boundaries within which their authority is exclusive." This Marshall decision argued that dependency does not negate internal sovereignty.

> A weak state, in order to provide for its safety, may place itself under the protection of one more powerful, without stripping itself of the right of government, and ceasing to be a state (Worchester v. Georgia).

The concepts of sovereignty and dependency were further elaborated in another Marshall decision, Cherokee Nation v. Georgia (1831). Indian tribes were referred to as "domestic dependent nations" whose unique relationship to the federal government resembled that of a ward to a guardian.

In affirming the "trust responsibility" and "guardian" role of the federal government, these court decisions established the plenary power of Congress over Indian affairs. But this power brought obligations. As explained by David Case in *Alaska Natives and American Laws*(2):

> [T]he ascendancy of the federal power over Native American communities creates an unequal *political* relationship upon which the Native Americans are compelled to rely. Their reliance requires generally that the federal government adhere to an "overriding duty ... to deal fairly with the Indians wherever located" (Morton v. Ruiz 1974) and "imposes a distinctive obligation of trust incumbent upon the

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> government in its dealings with these dependent and sometimes exploited people" (Seminole Nation v. U.S. 1942) (emphasis in the original).

The legal obligation to protect and provide for Native Americans has been acknowledged in a series of statutes, congressional appropriations, and court decisions. These are particularly important in the case of Alaska because Alaska Natives do not share with Indian tribes in the continental United States the same history of resistance, conquest, and treaty making with the federal government. In 1871, shortly after the U.S. purchased Russia's interest in Alaska, Congress ceased to make any further treaties with Native Americans. The statutes and appropriation legislation that have explicitly included Alaska Natives have clearly established that the "unique legal relationship", "trust responsibility", or "guardianship" as it is variously called, is not tied to reservation status and the federal government's management in trust of Native lands. Furthermore, they explicitly extend to Alaska Natives the same protection and services to which Native Americans elsewhere are entitled.

The principle of Native sovereignty has some important implications. Because Native American tribes and Alaska Native communities are considered sovereign entities, the relationship between them and the federal government is one of government to government. Programs and benefits to Native Americans are based on this unique political relationship, not on the basis of any racial criterion. Eligibility is not necessarily based on "blood quantum", i.e., degree of Indian ancestry, as is commonly supposed. Native status is based on recognition of a person's membership in a tribal entity or descent from a member. The tribe (in Alaska, a village or regional Native organization) as a political institution has the primary responsibility to determine its membership for purposes of participation in tribal elections, property distribution, and other rights arising from tribal membership. The federal government does not have the power to alter tribal determinations in these matters(3). Because of this unique, political relationship, federal programs that are exclusively for the benefit of members of a distinct ethnic group are not held to be racially discriminatory (see Morton v. Mancuri 1975; U.S. v. Antelope 1976).

This political relationship is also unlike other relationships between the government and its citizens whereby the government provides services for the needy. Natives, as Natives, are not wards of the state, nor is eligibility determined on the basis of individual need. However, as U.S. citizens, Native Americans enjoy a dual entitlement. They are eligible for social security programs such as Medicare and Medicaid in addition to their eligibility for benefits specific to Native Americans. This dual entitlement was reiterated by Congress in 1976 when it authorized IHS to receive reimbursement from Medicare and Medicaid for services provided to eligible Native Americans(4).

Another important principle is the distinction between federal services as privileges versus statutory entitlements. The former term refers to the view of services as gratuities that can be withdrawn at the discretion of federal administrators. Prior to a series of court decisions in the 1970s, benefits for Native Americans that were not tied to treaties and land cessions were considered "gratuitous appropriations" --revocable privileges--as opposed to entitlements. This was the case with appropriations for education, health, and other human services authorized under the Snyder Act of 1921 which could be revoked by an administrative officer such as the Secretary of the Interior (2). Supreme Court decisions in the 1970s (Board of Regents v. Roth 1972; Goldberg v. Kelly 1970; Pence v. Klepp 1976; and Morton v. Ruiz 1974) have replaced this conceptualization with the doctrine of statutory entitlements. This holds that benefits from federal programs are "expectancies" and they cannot be denied without fair hearings, rulemaking, and other due process procedures. As Felix Cohen writes in his *Handbook of Federal Indian Law,* "Existing statutory programs are so extensive that it is often appropriate to assume that a general duty to provide services has been undertaken(4)."

Congress does retain the constitutional power to limit or terminate services to Native Americans. In fact, Congress has historically limited services through appropriations insufficient to cover unmet needs. However, administrative discretion to restrict Native benefits is clearly limited by the "due process" considerations and the principle of statutory entitlements. Two Supreme Court decisions are particularly pertinent.

In Morton v. Ruiz (1974) the court ruled invalid BIA actions to deny general assistance to off-reservation Indians. Citing the "overriding duty of our Federal Government to deal fairly with Indians wherever located", the court ruled that the denial of benefits to those otherwise entitled Indians, without adherence to rulemaking provisions of the Administrative Procedures Act is "inconsistent with the distinctive obligation of trust incumbent upon the Government in its dealing with these dependent and sometimes exploited people". The court further concluded that:

> [A]gency power to make rules that affect substantial individual rights and obligations carries with it the responsibility not only to remain consistent with the governing legislation but also to employ procedures that conform to law. (Morton v. Ruiz)

In White v. Califano (1978) the issue was whether or not the federal government has a legally enforceable responsibility to provide Native health care, in this case mental health treatment. The court affirmed the federal trust responsibility as the basis of the court's jurisdiction in the case and cited the 1976 Indian Health Care Improvement Act as congressional recognition of that responsibility. The court concluded that:

> The Congress in 1976 stated that the federal government had a responsibility to provide health care for Indians. Therefore, when we say that the trust responsibility requires a certain course of action, we do not refer to a relationship that exists only in the abstract, but rather to a congressionally recognized duty to provide services for a particular category of human needs. (White v. Califano)

This interpretation of statutory entitlement to health and social service programs is particularly important to Alaska Natives due to the absence of treaties between Alaska Natives and the federal government.

THE FEDERAL GOVERNMENT AND ALASKA NATIVE HEALTH CARE

The relationship between Alaska Natives and the United States government began with the Treaty of Cession of 1867 wherein Russia transferred to the United States its interest in the scattered settlements along the Aleutian Islands, the Gulf of Alaska, and Southeast Alaska in exchange for $7.2 million. The treaty linked the treatment of Alaska Natives with that of American Indians by specifying that the "uncivilized tribes" of Alaska were to be "subject to such laws and regulations as the United States may, from time to time, adopt in regard to aboriginal tribes of that country."

Little was provided in the way of health care under the initial military occupation and the subsequent civil administration under the Organic Act of 1884. While a series of epidemics ravaged the Native population there were only a handful of physicians at military posts, on coastal patrol boats, and, starting in the gold rush era of the 1880s, at mission sites.

Since there was no Indian bureau in Alaska, federal funds for health and educational services were channeled through the Bureau of Education. Some appropriations were made specifically for Alaska Native programs, e.g., the reindeer herding program designed to transform Native hunters to more sedentary herders. In 1905 the Nelson Act authorized federal appropriations for education and support of Alaska Natives. This was the first of a number of statutes acknowledging federal responsibility for services specifically for Alaska Natives. Over the next twenty-five years, congressional appropriations to the Bureau of Education enabled the operation of health facilities either built or converted from school buildings at Nushagak, Nulato, Kotzebue, Nome, Russian Mission, Seward, Juneau, Sitka, Akiak, Noorvik, Unalaska, and Tanana.

The Snyder Act of 1921, a general assistance act for Indians, charged the Bureau of Indian Affairs (BIA) to direct, supervise, and expend congressional appropriations for "relief of distress and conservation of health" of Native Americans. It charged the BIA to employ physicians for that purpose. Although vague, the Snyder Act was the first legislation to specify government provision of Indian health care. Lacking BIA offices in Alaska, the act did not apply to Alaska Natives until 1931 when administration of Alaska Native affairs was transferred to the BIA from the Bureau of Education. From then on, education, health and welfare services were provided to Alaska Natives on the same basis as Native Americans elsewhere.

During these years when the federal government was taking responsibility for social services to Alaska Natives, judicial decisions and executive actions were simultaneously determining the federal-Native relationship regarding Alaska Native lands, subsistence rights, and the exercise of local governing powers. In 1932, the solicitor issued the following opinion on the status of Alaska Natives:

> From the foregoing it is clear that no distinction has been or can be made between the Indians and other natives of Alaska so far as the laws and relations of the United States are concerned whether the Eskimos and other natives are of Indian origin or not as they are all wards of the Nation, and their status is in material respects similar to that of the Indians of the United States. It follows that the natives of Alaska, as referred to in the treaty of March 30, 1867, between the United States and Russia are entitled to the benefits of and are subject to the general laws and regulations governing the Indians of the United States...(53 L.D. 593 (1932) "Status of Alaska Natives", quoted in[2].

In the years leading up to and following World War II, the federal government's responsibility for Native health care in Alaska was further confirmed through "legislation by appropriation", i.e., the demonstration of congressional intent through the expenditure of funds. Between the assumption of Native programs by the BIA in 1931 and the start of WWII when funds and medical personnel were diverted for the war effort, seven small hospitals were built in Alaska at Mountain Village, Kotzebue, Unalaska, Barrow, Bethel, Kanakanak, and Tanana(5).

In the postwar period, the Department of Interior commissioned two studies on health conditions in Alaska, the Barnett Report of 1947, and the 1954 Parran Report(6,7). The appalling occurrence of disease in the Native population reported in the studies prompted congressional appropriations for health research, medical facilities, field-based care, and village water and sanitation services, primarily to address the tuberculosis epidemic. The Parran group also recommended the transfer of BIA health programs to the Public Health Service. This was accomplished in 1954 under the Transfer Act which transferred the functions, responsibilities, and authority for Indian health programs from the BIA to the Public Health Service under the Department of Health, Education and Welfare. The creation of the Indian Health Service (IHS) was deemed advantageous in that it increased the medical personnel available for Native health care since the Public Health Service Commission Corps provided better career opportunities to physicians. The IHS was also in a better position to take advantage of the public health expertise and other resources of the Public Health Service and other HEW agencies.

THE ALASKA NATIVE CLAIMS SETTLEMENT ACT

The next significant legislation concerning the federal-Native relationship was the Alaska Native Claims Settlement Act (ANCSA) of 1971. While the act primarily addresses the question of Native lands, it is important for the discussion of health care since it has been suggested that ANCSA has somehow terminated the special federal-Native relationship and that the government is no longer obligated to provide human services to the Alaska Native people.

Alaska Native aboriginal land claims date back to 1867 when the Tlingits of Southeast Alaska protested the sale, from Russia to America, of the land they occupied. Tanana Athabascans asserted their claim to lands in Interior Alaska in 1912. In 1959, the Tlingit and Haida won compensation for lands taken from them. This judgement of the U.S. Court of Claims declared that the 1867 Treaty of Cession did not extinguish aboriginal title. Nor did the Statehood Act of 1958, which granted the state almost 104 million acres, settle the question of Native land rights. Section 4 of the Statehood Act disclaimed all rights in "any lands or other property, the right or title to which may be held by any Indians, Eskimos or Aleuts...or is held by the United States in trust for said Natives" (4).

With land selections imminent after statehood and with increasing efforts to regulate Native subsistence activities, the Natives' need for aboriginal title to lands and protection of subsistence became more urgent. A statewide organization of Natives, the Alaska Federation of Natives, was established in 1966. Its first joint effort was to request the Department of Interior to enjoin all disposals of federal land pending congressional settlement of Native claims. Secretary of Interior Stewart Udall imposed a land freeze in 1966 suspending patenting or approval of selection of public lands pending settlement of Native claims. Fortuitously for the Native claims movement, oil was discovered on the North Slope at Prudhoe Bay in 1968. In the interest of avoiding litigation that could delay North Slope oil development and construction of the Trans-Alaska pipeline, oil companies joined the state in pressing for a land claims settlement.

By the terms of ANCSA, signed into law December 18, 1971, Alaska Natives accepted forty-four million acres, to be selected from federal public lands, and $962.5 million in exchange for extinguishment of all Native claims based on aboriginal right, title, use or occupancy of the land in Alaska.

The act required the establishment of twelve regional corporations, one for each geographic region which encompassed communities of Natives who shared a common heritage. The regions approximated the areas covered by existing Native associations. The corporations were to own, and manage for profit, the land and its resources. The 76,526 Natives enrolled in the regional corporations were each entitled to 100 shares of stock in their regional corporation. Alaska Natives could also enroll in one of 203 village corporations created under the act to manage the surface rights to village lands (approximately half of the 44 million acres). (See (8) Arnold 1978 for a full description of the terms of ANCSA; and (9) Berger 1985 for an examination of its effects.)

ANSCA expressly extinguished aboriginal title to the lands ceded to the government. It did not, however, purport to affect federally provided health and social services to Alaska Natives. Sections 2(b) and 2(c) of the act express congressional concern for Native welfare and require that Congress accomplish a settlement "in conformity with the real economic and social needs of Natives" and that there be "maximum participation by Natives in decisions affecting their rights". However, the specifics of Native human service programs were left to the future recommendations of the Secretary of Interior who was directed by section 2(c) to:

> make a study of all Federal programs primarily designed to benefit Native people and report back to Congress his recommendations for

> the future management and operation of these programs within three years of the date of enactment of this Act.

The Department of Interior has yet to make such recommendations.

In addition to the ANCSA stipulation that there be "maximum participation by Natives in decision affecting their rights", there are judicial decisions of the early 1970s which have defined statutorily enacted federal services as entitlements, i.e., rights as opposed to gratuities. These decisions mandated that due process considerations were necessary for the denial of these entitlements. Thus, while Congress has the plenary power to withdraw entitlements, those already enacted cannot be administratively limited. They must be clearly, statutorily altered and this process must entail due process procedures and Native participation. ANCSA does not do this. There is no provision in ANCSA mandating termination of any federal programs.

POST-ANCSA LEGISLATION

Statutory enactments subsequent to ANCSA have specifically included Alaska Natives in legislation benefitting Native Americans. This legislation includes: the Indian Financing Act (1974); the Indian Self-Determination and Education Assistance Act (1975); the Indian Health Care Improvement Act (1976); and the Indian Child Welfare Act (1978). The Indian Self-Determination Act (Sect. 4) defines the persons to whom the act pertains as follows:

> (a) "Indian" means a person who is a member of an Indian tribe;
> (b) "Indian tribe" means any Indian tribe, band, nation, or other organized group or community, including any Alaska Native village or regional or village corporation as defined in or established pursuant to the Alaska Native Claims Settlement Act (85 Stat. 688) which is recognized as eligible for the special programs and services provided by the United States to Indians because of their status as Indians.

Specifically with regard to health care, the Indian Self-Determination Act and the Health Care Improvement Act mandate health care benefits and Native participation in the programs that provide these services. As the Indian Health Care Improvement act states:

> [I]t is the policy of this nation, in fulfillment of its special responsibilities and legal obligations to the American Indian people, to meet the national goal of providing the highest possible health status to Indians and provide existing Indian health services with all resources necessary to effect this policy.

CONCLUSION

Congressional statutes and appropriations, executive actions, and decisions of the judiciary have clearly and unequivocally expressed the special legal relationship between Alaska Natives and the federal government, and the continuing federal obligation to provide human services, specifically health care, to Alaska Natives. The Alaska Native Claims Settlement Act of 1971 has done nothing to extinguish this obligation. The Indian Health Care Improvement Act of 1976 strongly reiterated this relationship and the responsibilities it entails. The act explicitly states that the provision of health care to American Indian people, specifically including Alaska Natives, is a legal obligation of the federal government. Furthermore, the act declares it a matter of national policy and a national goal to provide "the highest possible health status to Native Americans.

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Special Feature

WITCH DOCTOR'S PROPHECY

Jaroslav Richter, M.D. (1)

It was the 28th day of December, and this newborn babe was not going to make it to the New Year.

The tiny baby, only a few hours old, lay on a pile of dirty rags in a one-room adobe house. Only occasional gasps hinted at life. The boy's body was bluish from lack of oxygen.

His father had come urgently through the night to the mission at which I was the new doctor. He had knocked timidly on my door. The old Guatemalan brujo--a witch doctor--had already told him his son was doomed to die: the Spirit had willed it so.

The father gave another timid knock, challenging the brujo's prophecy, his power, was to break a centuries-old tradition. But the Quiche Indian man was reaching for one last hope: that the physician from the outside world could help. Now he knocked with force.

As an intern at Los Angeles County General Hospital, I had become used to a frantic pace that included being jarred out of sleep in the middle of the night. But here, in the tiny, peaceful mountain pueblo of Novierro that knock seemed foreign.

It took me a while to wake up to the reality of an urgent sound in the Guatemalan night. I finally got up and opened the warped door of my old adobe building. There stood an Indian man dressed in the traditional garb of the Quiche--hat, red shirt, and white and black wool wraparound. His face was immobile, his eyes intense.

He stood with stoic dignity, but his voice quavered as he told of his newborn son, his son who was dying.

Doctors here were usually accepted skeptically. We were something to be humored but not trusted too deeply, and I resented that. Trust was reserved for the brujos, whose wisdom, handed from father to son, ranked with the deepest mysteries of life.

Still, the father stood there asking me to come. He had challenged tradition as he asked me to join him. I quickly woke up our two nurses, Carol and Randy, and we gathered our scant medical equipment. Then the four of us raced from our pueblo in the jeep.

At a sign from the father, we left the jeep and began to follow him along an uncertain path without lights. As we pressed forward my mind searched through the probably diagnoses, probing for what could go wrong in this newborn child, mentally testing and rehearsing the possible forthcoming steps in treatment.

After about 45 minutes of sometimes walking on narrow, mountainous paths breathlessly, through forests and across creeks, we finally arrived at the man's adobe house. It was a single room with no windows--only a door.

The smell of dampness greeted us inside. Wood smoke entered our protesting lungs and stung our eyes to tears. It took a few moments for our eyes to adjust before we could see what was happening in this dark, soot-covered room.

A small group of people huddled around the premature baby of the still baby barely handing on to its life. The signs of life appeared to drain as we watched. Only an occasional gasp was where the expected rhythm of breath should be, with irregular eye movements beneath closed lids.

His heartbeat could scarcely be felt, only 40 beats per minute, in contrast to a normal pulse of 160 strong, regular beats. He was blue in color from lack of oxygen, cyanosis betoken a fading life.

I fought against a feeling of resignation, the feeling of impotence, helplessness. Only in a major medical center, with an array of sophisticated equipment, specialists, and medicines, would there be a possibility of saving this baby. But we were far from anything like that; we lacked medicine and equipment.

When I completed my evaluation, I had to concur with the witch doctor. There was nothing to do to save this little life. Ready to leave, I turned to the mother and father and admitted by helplessness. "There is nothing I can do. I have no equipment. I can do nothing."

"But Doctor," Randy protested, stopping me on my way to the door. "You must do something."

While the parents watched, the nurse and I argued briefly in a language unknown to them, my medical knowledge pitted against her determination and idealism. I gave in. I picked the tiny bundle up and, with his parents, I carried him to our jeep, knowing that would placate Randy, but, nonetheless, fearing that I was right.

On the way back to our quarters I mused about the three of us. Only a few months ago we "health professionals" had arrived in Novierro, fresh from our intern-ships at Los Angeles County Hospital. Carol Thompson and Carolyn "Randy" Randall were registered nurses. I, a doctor.

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(1) 1200 Airport Heights, Suite 340, Anchorage, Alaska 99508

While working in that vast, cosmopolitan medical facility, we had dreamed of broadening our medical horizons, of sharing medical knowledge and experience in a new environment and culture. To do so, we searched out a sponsor from the Orange County Medical Association, and we even collected Blue Chip and Green Stamps to raise funds. Now here we were in the remote Guatemalan village of Novierro, helping Catholic missionaries from the State of Washington.

The instant we arrived, our quarters became the hospital. Our quarters consisted of a rickety bed and chair, a small oil stove, and a few primitive wood benches. An ancient warped door kept the wind from blowing into the room.

I had just been in a hospital where my medical knowledge was challenged daily, but I was not ready for this baby. I felt helpless and feared what would happen.

I tried to organize my thoughts, but all of my knowledge seemed to require the sophisticated equipment of the medical center. Nonetheless, I knew I must make the effort to provide some kind of scientific treatment.

The baby looked calm, so still. Too still. Then he would shudder and gasp for air. His skin was now even more cyanotic, or darker blue from the lack of oxygen.

What was gong one? What was the cause? What was the cause of the problem? Was this something I knew but just didn't recognize, or some kind of new and exotic, but horrible unnamed disease, or something endemic to this particular area, unknown yet to me?

That was it. I certainly didn't know, and perhaps I was also afraid to speculate too much. I was afraid to make decisions. The wrong choice, the wrong decision could terminate this already faltering spirit.

I could not stall any longer, inert. Some decision had to be made. Any decision, but some decision had to be made. Some kind of treatment had to be initiated.' Now the boy was not breathing, he was only gasping in his terminal effort to sustain life. We started mouth-to-mouth resuscitation, forcing breath into and out of his lungs repeatedly, over and over, continuously taking turns between the three of us.

But as we worked we knew fear. Some diseases can be transmitted mouth-to-mouth. Should that little baby have meningitis, it could be transmitted, and all of us could die.

We attempted to do a spinal tap in consideration of possible meningitis, however, lacking the right equipment we gave it up. Instead, we put our trust in the hope that the boy did not have some dreadful disease.' I realized that his little body must be dehydrated. It had not been receiving any outside food or fluids, and he needed calories to sustain his energy and keep his little heart beating. There must also be some acidosis, since his lungs were not expelling gas as the product of metabolism in his body.

We had no intravenous fluids to administer. What else could be done?

Our scientific measuring device was a teaspoon. We boiled water, then put baking soda and honey into the solution after I had calculated the proper proportions for his body mass. The water would take care of the dehydration, the honey would provide calories, and the baking soda would counteract the acid condition in his system.

To administer our preparation so the baby would not choke, we used an old urinary catheter, likewise boiled. We inserted the catheter gently into the child's mouth and on into his stomach. Through the tube small amounts of our solution were slowly delivered at a rate his system could absorb.

At the same time, with the catheter inside him, we tried to maintain the boy's breathing. We continued with this for a few hours, but the heart showed a more erratic, slower pattern until it stopped.

We applied CPR to his tiny body until we obtained a particular rhythm. Then we tried again to follow a certain sequence of mouth-to-mouth resuscitation, this time with cardiac massage, but after a while we abandoned our efforts for regularity and scientific precision with the rhythm. The best we could do was to give the squeezing of the chest some pattern, though certainly not precise.

Among our limited stock of drugs, most of them very elementary, we did have one vial of Epinephrine. We injected it directly into the heart, although at that point it seemed to do very little.

So we continued external cardiac massage. Over and over, monotonously, we just continued, switching between the nurses, who had fervor, hope and enthusiasm, and I, who had been pessimistic about the outcome from the beginning.

I worked with intensity, and with professional dedication, but certainly not with any clear faith or belief that the boy we were struggling over would survive. I continued to work out of feeling for the two young nurses who were with me, two idealistic, totally unrealistic nurses.

One day went by. Then the second night passed without giving us any hope that the trend would reverse. If anything, the small child seemed to slowly deteriorate. His death seemed inevitable.

Indeed, there was rising respect for the witch doctor's wisdom in saying that the sickly child should be left to die. I began to feel like a fool in defying the old wisdom, in defying nature, in choosing to interfere with the traditions of the village.

We continued mouth-to-mouth resuscitation. At long last, 3 days later, the heartbeat appeared to be normal. Perhaps external cardiac massage could be terminated. At this time, we did and the heart continued to beat on its own.

Aside from the heartbeat though, there were no other signs of more life in that little body, which appeared more and more like a limp little wet rag than a natural living infant.

The third night came, New Year's Eve. In a strange combination of fatalism and faith, two of us took the jeep, and went to attend church services while one nurse continued mouth-to-mouth resuscitation. when we came back to our makeshift hospital, we continued the same rhythm of mouth-to-mouth respiration, but with a growing sense of fatalism and a shrinking sense of faith.

It was 12 o'clock New Year's Eve, the beginning of a new day and a new year. As the first minutes went by, our rhythm of breathing continued. At five minutes after the hour, a sign of life appeared: the heart began to beat regularly, strongly, with its normal rhythm.

Then there was a resistance to the forced breathing. The infant was breathing on his own! The blue little body was now pink. It was quivering. It was moving. It showed signs of life.

Eulalio was indeed living. All our efforts had won over the greedy fingers of death. All the efforts to sustain life had won over the witch doctor's prediction that the boy would die. The application of modern science had won over superstition.

The child now showed strong efforts to sustain its own life. He had won. Eulalio lived and became stronger through the efforts of us, the foreigners. We had won over the witch doctor.

Carol and Randy's faith that something could be done, and their faith that I too, could do it, had dragged me along, and together we had given a New Year's present to Eulalio.

It took us a while though, to realize that he had given a New Year's present to us as well. Through Eulalio, the Indians of the area knew we had proved the witch doctor wrong, and they began to trust us. And with that trust, this little baby gave us the real beginning of our practice in the mountains of Guatemala.

Eulalio is now twenty years old, and even though I am in Alaska I occasionally hear from him. He is a fine, healthy man.

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References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
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4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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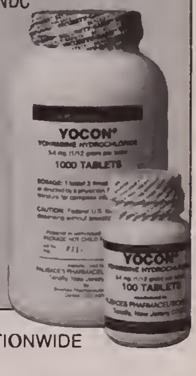

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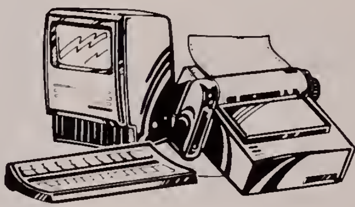
We lease anything from a to a  or even a . Just give us a .

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ALASKA PUBLIC HEALTH ASSOCIATION

President's Report
June, 1990

by Brian Saylor, Ph.D., M.P.H.

The Alaska Public Health Association Annual Meeting and Banquet were held in Whitehorse, Yukon Territory in conjunction with the 8th International Congress on Circumpolar Health. There appeared to be a greater turnout for the Annual Meeting than the association typically has when meetings are held on Alaskan soil. While an exact count was not kept (there were a lot of members and observers who drifted in and out) we estimated that about 45 to 50 attended the meeting. Along with the routine annual business, the Association discussed and passed seven resolutions and a position paper on the need for arctic health and medical research.

ALPHA welcomed the incoming Board members. Bill Dann was elected President-elect of the Association. Bill, known to most of the members, has extensive experience in Alaska health issues. He served as Director of the Norton Sound Health Corporation for many years and now runs his own consulting firm in Anchorage. A roster of the members of the ALPHA Board and their committee assignments is included in this edition of the newsletter.

The ALPHA Banquet was a huge success. Over 70 people attended. We had representatives from Denmark, Greenland, Siberia and Canada in addition to our own membership. Following a 10-course Chinese buffet, we listened to two distinguished speakers discuss how professional associations can help advance arctic health policy and research. First, Ann H. Wieler, Project Officer of the International Health Secretariat of the Canadian Public Health Association, commented upon the cultural considerations in providing health services to northern people. Dr. John Middaugh's speech followed, recounting his experience in working with professional associations, including ALPHA, on arctic health research for the past 10 years.

The Association presented six awards in recognition of the outstanding efforts of Alaskan health care professionals. They are as follows:

ALPHA Barbara Berger Award	Thomas Nighswander, M.D.	Anchorage
Alaska Meritorious Health Service Award	Bea Shepard	Juneau
Alaska Health Achievement Award	James Berner, M.D.	Anchorage
ALPHA Long Term Service	Carl Hild	Anchorage
ALPHA Annual	Corlis Taylor	Bethel
Alaska Community Service Award	Alaska Health Fair	

I look forward to working with you this year, continuing to identify and work towards public health goals in Alaska. I encourage each one of our members to participate in ALPHA by sharing ideas, opinions and energy which can be directed toward the achievement of these goals.

Alaska Public Health Association
Proposed Position Paper A - 90

Advocating for Organizational Changes in Federal Agency Infrastructure to Support an Arctic Health Research Agenda

Problem Statement:

There is a growing awareness of the opportunities in arctic health research throughout the federal government and the nation's health research community. In order to effectively pursue a comprehensive arctic health research agenda, significant changes must be made in the federal government agency infrastructure. This position paper outlines the changes that the Alaska Public Health Association considers essential to the success of an arctic health research policy.

Background:

The health research community has watched with great interest the development of arctic health research under Arctic Research and Policy Act of 1984 (ARPA). Many individuals and organizations have contributed to this effort and remain committed to its sustained growth and success.

There have been several notable successes, albeit modest, as a result of ARPA. The increased activities occurring between Alaska and the Soviet Union have been well publicized. Less well known are the increased efforts of the State of Alaska, Department of Health and Social Services, to collaborate on a broad array of health topics with the Canadians. In addition, the involvement of American health scientists in the 8th International Congress on Circumpolar Health, Whitehorse, Yukon, Canada in May 1990 is expected to be greater than at any time in the past. Recently, Alaskans have participated in major international arctic conferences on Tobacco and Health, AIDS, and Meningitis. Plans are underway for arctic conferences on Tobacco and Health, AIDS, and Meningitis. Plans are underway for future international workshops on Echinococcus and Injury Control. The World Health Organization has indicated an interest in and support for establishing an Arctic AIDS Regional Resource Center.

These achievements and successes are now being followed by new initiatives. Some of these new activities in health research have begun in Alaska. The Indian Health Service has obtained new funds to help support the Alaska Native Cancer Registry for five years. The Alaska Chapter of the American Cancer Society has been joined by the Alaska State Medical Association and the Health Care Coalition of Alaska to advocate for a statewide cancer registry. The University of Alaska is actively working to develop new health research projects, and the WAMI program is emerging as an important resource.

The Arctic Health Research Agenda

A consensus research agenda is already developed. The health community is united as to the topics that need to be studied(1-3). The work of the past 5 years certainly does not need to be repeated nor does the existing research agenda need to be reinvented. The four major research areas in the Annual Report of the USARC are valid today. Continued support should be given to:

1. Injury Control Research
2. Cancer
3. Diet and Atherosclerosis
4. Infectious Disease

The highest priority now is to put solid meritorious, peer reviewed projects in the field. The Alaska Public Health Association believes that health research must meet the following criteria:

- Research important to national goals and objectives
- Peer-reviewed
- Evidence of benefit to arctic people
- Evidence of cooperation between federal and state agencies
- Research neglected in past
- Able to be accomplished with enhanced resources

Organizational Changes Needed to Support the Research Agenda

Given that there is an agreed upon, defined, and broadly supported agenda of arctic health research topics, how can we get the research agenda implemented?

It is the position of the Alaska Public Health Association that the following organizational changes be pursued to implement the arctic health research agenda:

1. There should be increased financial support for the Polar Research Board and from the Federal government's Department of Health and Social Services (DHSS).

2. An Arctic Desk in DHSS or NIH or CDC should be established.

3. The mission of CDC in Alaska should be redefined.

These are discussed in detail below:

1) INCREASE FINANCIAL SUPPORT FOR POLAR RESEARCH BOARD AND DEPARTMENT OF HEALTH AND SOCIAL SERVICES

Through the years, the Polar Research Board (PRB) has not received funds from DHSS to support its work in health-related areas. In order to enable more effective and sustained efforts in developing polar health research, the PRB will need core funding support funds from DHSS. This has compromised the ability of either organization to assume a strong leadership position on arctic health issues. Due to this situation, arctic health research fell into a crevasse between Division of Polar Programs (PP) and DHHS. Those approaching DHHS were told that arctic health was polar research and therefore a mission of the Division of Polar Programs. Those approaching the Division of Polar Programs were reminded that DPP is part of the Division of Geosciences and that DHSS funds health research. Because DHHS never established a specific polar or arctic mission, arctic health research fell into 2onethis crevasse with, for many years, no rescuer nearby. Increased funding can help resolve this situation by clarifying the responsibility for arctic health research in the PRB.

2) ESTABLISH AN ARCTIC DESK IN DEPARTMENT OF HEALTH AND HUMAN SERVICES

In "The National Arctic Health Science Policy" report (1), the American Public Health Association called for the creation of an arctic desk within DHHS. Others have done the same. The location of this new office could be in the National Institutes of Health or the Centers for Disease Control, but would be even better located in the Office of the Assistant Secretary for Health . This office should be headed by a scientist (with a M.D. or Ph.D.) and should be responsible for coordinating the implementation of arctic health research.

3) REDEFINE THE MISSION OF THE CENTERS FOR DISEASE CONTROL IN ALASKA

At this time, the only dedicated federal health research unit in the arctic is the Arctic Investigations Program (AIP), Center for Infectious Disease (CID), in Anchorage, Alaska. However, this agency is but one program of the many Centers within Centers for Disease Control. Because the mission of the Arctic Investigations Program is primarily to address infectious diseases, they are limited in their ability to conduct research into other arctic health problems.

As the State of Alaska grows, priorities and agency responsibilities must be constantly reevaluated so that essential services are maintained and new or emerging problems anticipated or recognized.

We recommend that CDC expand its arctic mission to include all priority research areas with special attention to alcohol and illicit drugs and injury prevention.

Summary:

Arctic health research is continuing despite significant organizational problems. An accepted research agenda can be successfully pursued by advancing clear organizational and funding changes. The Alaska Public Health Association recommends that these changes be implemented by the Federal government to ensure the conduct of effective and relevant arctic health research for the benefit of all Alaskans and other arctic and sub-arctic people.

Actions:

The recommendations contained in this position paper should be forwarded to affected agencies.

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1. "The National Arctic Health Science Policy," American Public Health Association Report Series, American Public Health Association, Washington, D.C., August, 1984.
2. United States Arctic Research Plan, InterAgency Arctic Research Policy Committee, Washington, D.C., July, 1987.
3. Entering the Age of the Arctic, Opportunities and Obligations of an Arctic Nation. Report of the U.S. Arctic Research Commission to the President and the Congress of the United States of America. (For the Period 1 October 1986 - 30 September 1987.) January 31, 1988.

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History of Medicine in Alaska

The Alaska Railroad Hospital and Its Last Superintendent, Fred Braun

Fred Braun came to Alaska in January 1934. After graduating from the University of Washington in pre-med, he was accepted at the University of Virginia Medical School. Since the Great Depression was at its deepest, however, he could not afford to go. Because he had studied to be a laboratory technician at the Virginia Mason Clinic during his university days, he came to Alaska on a one-year contract with the Alaska Railroad to serve in this capacity at its base hospital in Anchorage.

At the end of the contract, he went back Outside but was so overwhelmed by the rat race that he returned to Alaska. Actually, he was only away for one month, half of which was spent sailing from Seward to Seattle and back.

He worked primarily as a lab tech but often doubled as an orderly, bathing "old fellows" in what was probably their first bathtub encounter in years(1). In the latter part of 1935, Braun became acting superintendent, then superintendent, when Dr. Earl Albrecht, scheduled to fill the position, was sent to the Matanuska Valley to practice instead.

Construction of the Alaska Railroad Hospital was started on August 12, 1916 and completed on December 1. It cost approximately $35,000 and was the largest as well as the best equipped in the Territory. A frame structure 42 by 100 feet, with two stories, an attic and a basement; it could serve fifty patients.

It had been placed on elevated ground on 3rd Avenue between A and B Streets. Proudly the newsletter, Alaska Railroad Record, noted that the hospital was "far enough removed from the business section of the town to insure insulation from activity and noise-so essential to the welfare of the patients."

It contained an X-ray room and a morgue, as well as four sleeping rooms for employees in the attic. In addition, each floor had been provided with ample bath and toilet facilities, hot and cold water, steam heat, and electric lights. Fred Braun notes that the elevator was hand-operated by pulling on a rope.

The Anchorage Hospital was responsible for supplying and supervising the field hospitals at Potter Creek, Talkeetna, Matanuska and King's River. These were visited periodically by Commission physicians "not only in the interest of the health of the railroad employees but also for the purpose of seeing that sanitary regulations, as prescribed, are being observed."

The railroad had been built from Seward to Turnagain Arm by private enterprise. During World War I, when Alaska seemed vulnerable, the federal government had purchased the 70.8 miles of track and assigned the task of completion to the Army Corp of Engineers. To avoid the problem of unionization, the Alaska Engineering Commission was created to contract out the construction to various groups. The task was supervised by the Corps of Engineers but the Commission was in charge.

Because the Alaska Railroad Base Hospital was the only one in the area with first class conveniences and equipment, an executive order in June, 1916 permitted the hospitalization of patients other than railroad or government employees.

During the influenza epidemic of November 1918, the residents of Anchorage were first hit by the disease, but later the construction camps were overwhelmed. Many of these patients were brought to Anchorage for treatment in the hospital.

In 1939 Providence Hospital opened and admission of private patients was discontinued except for those cases where the Railroad Hospital had equipment essential for treatment not available at Providence.

In the mid-thirties, when Fred Braun became superintendent, he became jack-of-all-trades. He served as X-ray technician, pharmacist and anesthesiologist, all self-taught. He ordered all medical supplies as well as the year's food supply for the entire railroad system. These came by ship from Seattle, were transferred to the railroad in Seward and taken to Nenana where they were further distributed by river boat. The last boat down the Yukon would leave extra medical supplies with the Catholic Mission at Holy Cross. Each year there was a question as to whether the river boat would get back to Nenana before the freeze or pull over into a slough for the winter.

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(1) Dr. Rodman Wilson recalls an "old fellow" who came in from the Bush to Old Providence Hospital in 1959 for cataract surgery by Dr. Milo Fritz. He was so dirty that the sisters gave him a bath. He promptly caught pneumonia. When asked later when he had last had a tub bath, he replied, "Chicago, 1903.

In the spring, Fred did the annual physical examinations for all employees. The criteria for passing were "if warm and could walk into the room." He notes that in Seattle at Virginia Mason Clinic men were old at sixty, while in Alaska, many seventy and eighty year olds were able to pass the railroad physical and work hard all summer. He attributes this to their demanding lives as prospectors.

Dr. Joseph Romig was the chief surgeon; Dr. A.S. Walkowski, the railroad surgeon. Both had offices in the hospital and anything of a serious nature was referred to them. For a while they were the only doctors in town.

Late one night Dr. Romig called Fred because there was a problem in Talkeetna. They mounted a gas powered Ford chassis on steel wheels with two seats along each side. An engineer drove the vehicle all night at 20 miles per hour with Dr. Romig and his cigar, Fred, and Frank Hawkman, a deputy marshal, arriving in Talkeetna at 6 a.m.

"Where is the prisoner?" asked the marshal. "How's the patient?" Dr. Romig asked. "If he has lived this long, he will live through breakfast." So they trooped off to the Roadhouse to eat, then to a cabin on the Susitna River where they found a white torso on a dark mattress on the floor. The man had had an argument with his brother-in-law who, in rage, had cut him from the right mastoid, across the back of his neck and around to the right nipple. He had been covered with flour to stop the bleeding. When Dr. Romig asked how he was feeling, the patient replied, "all right." The ghastly slash hit no major arteries or nerves. The mattress with patient was placed on the gas car, and back they went to the hospital where the patient was cleaned and stitched. The brother-in-law was sentenced to 5 years in prison. After completing his sentence, he borrowed $5 from the marshal and was not seen again.

The Matanuska colonists traveled from Seattle by Army transport ship. During the voyage, an epidemic of scarlet fever broke out. Upon arrival in Alaska, to combat this contagious disease, an isolation ward was opened in a house near the hospital. Since there was a limited staff, mothers had to take care of their children. Food, bedding and medicine were furnished. Fred remembers the quarantined adults and children looking pensively outdoors from the windows.

No sooner however had the Superintendent closed the isolation house and drained the water, when he got scarlet fever. Dr. Walkowski gave Fred a shot which made him very sick. He decided that the treatment was worse than the disease but he recovered from both.

In October of 1937, there was an explosion in the Evan Jones Coal Mine at Jonesville. Fourteen men were killed and many were injured. A special train carrying doctors and nurses was sent and returned the injured to the hospital. The railroad depended upon this mine for coal.

Two covered, seven-foot wide ramps were built at the hospital. They descended from the second floor to the ground and were intended to provide emergency exit for patients in their beds.

At the beginning of World War II, the Army used the Railroad Hospital until its own building was completed at Fort Richardson in May 1940.

The Railroad Hospital had to refuse private patients when the Army took over. It also provided the new Army hospital with medical supplies. These were loaned and were replaced item for item without exchange of money.

Because everything came by ship, the system was often jeopardized by frequent strikes by longshoremen. Once during the late thirties Railroad Superintendent Olson had gotten army ships to bring supplies.

The Railroad Hospital took care of other agencies, such as the Alaska Road Commission, without being funded by them. It had a contract with the Bureau of Indian Affairs for care of Natives around Cook Inlet as well as some in the interior at Ruby and Flat. Miners at the Fern and Independence Mines were treated on a fee basis.

Laundry from the hospital was done at the Curry Hotel 114 miles away. It was sent dirty on a northbound train and returned clean on a southbound train. This meant having a lot of linen.

Fred met his wife when her father was hospitalized at the Railroad Hospital. She taught school for the BIA at Unalaska, sailing on a small motor schooner between Seward and Unalaska. It was a rough trip at best, but especially so in December during the Christmas tree drop when the Star stopped at many villages.

Dr. Heitmeier succeeded Dr. Romig as Railroad Surgeon but soon left because of the war. There were wild rumors that a Japanese carrier was in Cook Inlet which was followed by the invasion of Attu.

Dr. Works was an EENT specialist who was sent a cross-eyed pregnant woman. After her arrival, however, he hurriedly boned up on OB and, with the help of a nurse, delivered the baby. Later, he corrected her eyes too.

Dr. George Davis became chief surgeon. He had served in Malaysia, even treating the royal family, and after retirement had come to Alaska to serve as cannery doctor in Bristol Bay. He joined the Railroad Hospital and stayed until it closed in 1950. The last few years part of the building was an outpatient facility, the rest was used as a dormitory for workers. This was affectionately known as "The Anchorage Hilton."

During the mid thirties, Dr. W. B. Shore had a "little hospital" on the western end of town. He took care of the "girls" who got in trouble "on the line." His nurse/wife managed the office and nursed the patients. The "girls" came to the Railroad Hospital once a month for a health check. Fred Braun remembers one in particular who was charged $10 for a chest X-ray. After receiving the bill, she remarked, "Jesus, Boy, you've got a better racket than I have."

When Fred Braun got tuberculosis in 1941, he spent three months in bed during which time Edith Baily Karterman took his place.

A doctor in Kotzebue had a theory that the Natives were swallowing caribou hairs. He requested that the appendices he sent be checked for animal hairs. Hairs were found in a few of them. But this is a story without a point since nothing ever came of it.

After the final closure of the Railroad Hospital on February 29, 1960, it was sold for $500 and finally torn down in 1962.

Fred Braun stayed on to manage the Railroad Medical Association which was a plan to provide medical coverage for railroad employees. A fund comprised of deductions from wages was used to pay physicians and pharmacists. Later instead of increasing wages, the Railroad added families to the plan.

Fred retired in 1968.

He has fond memories of the personnel of the Railroad Hospital, such as Esther McElligott, Esther Wolfe, and Ruth Dodd. There was one practical nurse, Emily Craig who had married a Finnish carpenter, Arthur Craig. In 1898, they drove a wagon from Chicago through Canada to the McKenzie River where they wintered. "Craigie" nursed in the campgrounds of the Porcupine and Yukon River. They lived in Nome for several years. He eventually went to work for the railroad in Anchorage and she at the hospital doing practical nursing, making bandages and mending linens. After the deaths of her husband and of Mrs. Joseph Romig, she married Dr. Romig.

Fred has devoted himself to the Anchorage Cemetery, identifying graves and recording its history. A mystery is a man who has two separate graves.

The Railroad Hospital was an important chapter in the medical history of Alaska.

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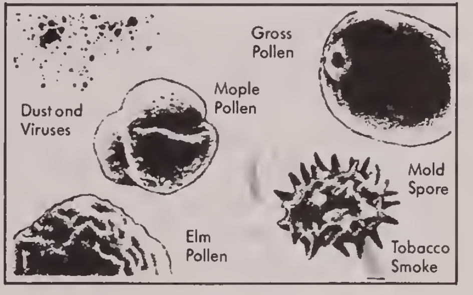

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American Society for Circumpolar Health

Summary of the VIIIth International Congress on Circumpolar Health

Approximately 750 delegates from 15 countries attended the Eighth International Congress on Circumpolar Health held at Whitehorse, Yukon, during the week of May 21 - 25, 1990. Dr. Brian Postl, President of the Canadian Society for Circumpolar Health, was also President of the Congress. The Scientific Program Committee with Dr. Kue Young as Chairperson structured the program. The International Advisory Committee and the Yukon Steering Committee along with the residents of Whitehorse itself, and many others, all contributed in coordinating and orchestrating a most successful congress.

There were over 420 papers for oral and poster presentations. These were mainly grouped in the categories of: mental health, infectious disease, oral health, nutrition, chronic diseases - cancer, diabetes, cardiovascular, injury, environmental, health care of mothers and children, of the elderly and disabled, occupational health, cold physiology and human biology, and health care delivery.

This year was the first time the Congress had broadened to include sessions focusing on the health needs of indigenous peoples and with presentations by Native health practitioners. The topics ranged from traditional Native medicine, indigenous control of health services to health education in the villages and professional development.

Invited speakers at the plenary sessions included Thomas Berger, Robert Fortuine, Emoke Szathmary, Ted Mala, R. Baruzzi, and Yuri Nikitin. There were also six workshops or meetings of working groups held: Circumpolar Cancer Study Group, USSR - Canada Transarctic Skitrek, SCAR (Scientific Committee on Arctic Research) Working Group on Human Biology and Medicine, SCAR Ad Hoc Group on Antarctic Space-related Human Factors Research, Viral Hepatitis, Otitis Media and Hearing Loss.

At the closing banquet, the Jack Hildes Memorial Award was given to five individuals honoring their long standing contributions in health research in the circumpolar regions. Dr. Frederick Milan, Professor Emeritus at UA - Fairbanks, was one of the recipients for his work in cold physiology among Eskimos. Dr. Milan, along with Dr. Earl Albrecht of Alaska, was instrumental in the development of the circumpolar conference. Other recipients of the Jack Hildes Memorial Award were Dr. Henri Forsius from Finland; Dr. Otto Schaeffer from Edmonton, Canada; Dr. Yuri Nikitin from Novosibirsk, USSR; and Evelyn Campbell, a nurse from Labrador, Canada.

The International Union for Circumpolar Health was held on the last day of the congress. J.P. Hart Hansen was elected President, Carl Hild of Anchorage, Alaska and treasurer of the American Society was elected the new Vice-President, and Kue Young was elected Secretary. The American Society for Circumpolar Health President John Middaugh presented plaques in recognition of their efforts for assembling the Congress to J.P. Hart Hansen (Pres - IUCH), Brian Postl (Pres - CSCH), Doug Bell (Secretariat General - ICCH), and Kue Young (Scientific Session - Chair).

The reasons for holding a congress every three years are: 1) to bring together medical scientists, health care delivery specialists, health administrators and health consumers to discuss the state of the art in their respective fields, 2) to allow national and international participants to observe and discuss the health situation in their own countries, and 3) to relate solutions to health problems in other parts of the world to the unique problems of circumpolar regions. Symposiums on circumpolar health were held in Fairbanks, Alaska (1967), Oulu, Finland (1971), Yellowknife, C.N.W.T. (1974), Novosibirsk, USSR (1978), Copenhagen, Denmark (1981), Anchorage, Alaska (1984), Umea, Sweden (1987), and Whitehorse, Yukon. The next two symposia are planned for Novosibirsk, USSR in 1993 and Anchorage, Alaska in 1996.

ASCH Breakfast Meeting

The American Society for Circumpolar Health (ASCH) "Get Acquainted" breakfast for all U.S. delegates and guests attending the VIIIth ICCH in Whitehorse, Yukon, was held at the Westmark Klondike Inn on Wednesday, May 23, 1990.

The no host buffet breakfast was attended by 54 members and guests, who braved the sudden changes in the weather during their walk and rides from their respective lodgings. All enjoyed the opportunity to get acquainted with our non-Alaska members and guests. There was good representation from Alaska, Canada and the "South 48".

Outgoing President John Middaugh served as the master of ceremonies. He outlined a few of the accomplishments of ASCH in the past year and the challenges facing the Society. The biggest challenge will be preparing to host the Xth ICCH in Alaska in 1996.

In recognition of long service to the Society, plaques were presented to outgoing officers John Middaugh (Pres.), Robert Fortuine (VP), Helen Beirne (Treas.), and Frank Pauls (Sec.).

IXth International Congress on Circumpolar Health

The IXth International Congress on Circumpolar Health (ICCH) will be in Novosibirsk, USSR during June, 1993. Start making plans now to attend this Congress.

International Union for Circumpolar Health (IUCH) Council President J.P. Hart Hanson announced the 1993 Congress site to the IUCH General Assembly meeting during the VIIIth ICCH in Whitehorse, Yukon, Canada. The Council had accepted the Siberian Branch of the USSR Academy of Sciences, Medical Section's invitation to host the IXth ICCH, during June, 1993, in Novosibirsk, USSR.

Further details regarding housing, transportation and specific dates will be announced in the near future by the Organizing Committee for the IXth ICCH.

All members of the American Society for Circumpolar Health will be advised of details as soon as they are available.

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PROGRAM NOTES
ARCTIC INVESTIGATIONS LABORATORY
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PROTECTION PROVIDED BY HEPATITIS B VACCINE AT 7 YEARS AFTER THE FIRST DOSE OF VACCINE

Several studies have demonstrated the immunogenicity and efficacy of hepatitis B vaccine in preventing infection with hepatitis B virus (HBV). Further studies have found that the protection provided by hepatitis B vaccine lasts for at least 5 years from the first vaccine dose. This report presents data indicating that in a Yupik Eskimo population hyperendemic for HBV infection the protection provided by hepatitis B vaccine persists for at least 7 years.

In 1981, a hepatitis B vaccine demonstration project was conducted in southwest Alaska, a population hyperendemic for HBV infection, to determine the immunogenicity and efficacy of the hepatitis B vaccine. 3,988 Yupik Eskimo residents of 17 villages were screened for HBV markers and 1,630 susceptible persons were immunized using the recommended three dose regimen of plasma-derived hepatitis B vaccine (Heptavax, Merck, Sharpe & Dohme, Rahway, NJ) by intramuscular injection. Teams visited each study village annually for 7 years to obtain blood specimens from the study participants. Sera were tested for markers of HBV infection - hepatitis B surface antigen (HBsAg), antibody to hepatitis B core antigen (anti-HBc), and antibody to hepatitis B surface antigen (anti-HBs).

One year after receiving the first dose of vaccine, 95 percent of vaccine recipients demonstrated anti-Hbs levels of 10 International units (mIU) or greater, an antibody level considered adequate to confer protection from significant HBV disease. Persons achieving anti-HBs levels of 10 mIU or greater are considered to be responders to the hepatitis B immunization. The proportion of immune persons responding to vaccine with an anti-HBs level 10 mIU or greater at 1 year was lower in older persons than younger.

After 7 years of follow-up, 26 percent of those initially having an immune response to vaccine of 10 mIU or greater, subsequently had anti-HBs levels less than 10 mIU. Anti-HBs levels in responders at 7 years were higher in younger persons than in older persons and higher in those initially having high levels. The median anti-HBs level has declined from a high of 307 mIU at 1 year after the first vaccine dose to 36 mIU at 7 years. During the 7 years after first doe of vaccine, five of the responders and two persons with an anti-HBs response less than 10 mIU developed anti-HBc and boosted their level of anti-HBs (Table). One additional person developed anti-HBc at 12 months after the first vaccine dose and may have been infection at the time of the third dose. Although infection had occurred, none developed hepatitis B surface antigen or had clinical hepatitis.

Our data indicate that the risk of HBV infection to most persons with an initial anti-HBs response to HBV vaccine of 10 mIU or greater is low regardless of the initial antibody level attained.

COMMENT

This study extends the information provided in an earlier report of the 5-year follow-up of this population hyperendemic for HBV infection. During the intervening two years median anti-HBs levels continued to decline. The proportion of protected persons declined to 74%. Three additional anti-HBc-positive infections were detected, one in a nonresponder, for a total of eight infections in 7 years. The annual incidence of infection in the study population is 0.85/1000, down from 50/1000 prior to immunization. This study continues to indicate that hepatitis B vaccine provides a high level of protection from adverse HBV events (symptomatic hepatitis and the development of the chronic carrier state). Though occasional infections do occur, they are asymptomatic and, as determined by currently available tests, have not been HBsA-positive. These data suggest that protection against significant HBV infection lasts for at least 7 years and probably longer. We do not see a need to recommend routine booster immunization in this study population at this time.

Follow-up of this group of vaccinees is ongoing and results of anti-HBs levels and evidence of infection will be available to assist in determining when booster doses may be needed.

For additional information contact Dr. Brian McMahon, (907) 279-6661 or Dr. Robert Wainwright, (907) 271-0411.

TABLE

Age[1]	Sex	First[2]	Anti-HBs level (MIU)		
		anti-HBc positive	Highest prior to infection	One year prior to infection	At time of 1st anti-HBc-pos.
22	F	12	21[3]	na	214[4]
55	F	24	4	4	604
45	M	48	505	173	176
46	M	60	36	0	208
1 8/12	M	60	1120	54	182
46	F	72	44	1	1424
1 4/12	F	84	1011	10	539
45	F	84	2	NA	229

[1] at first vaccine dose
[2] months after first vaccine dose
[3] level at the time of third vaccine dose (6 months prior to first anti-HBc-positive)
[4] anti-HBs level in sample ratio units (SRU)
na not available

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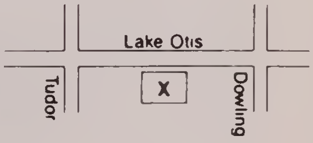

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GLIMPSES OF ALASKAN MEDICAL HISTORY

Edited by Robert Fortuine, M.D.

A NASTY CASE OF FROSTBITE ON KODIAK ISLAND (1808)

Archibald Campbell was born near Glasgow in 1787 and as early as the age of 13 went to sea as an apprentice. Over the next six years he sailed on five different ships, largely in the West Indies trade. In 1806 he signed on with the *Thames Indiaman* en route to China. At Whampoa he was induced to desert by an offer of better wages and joined the crew of an American ship *Eclipse*, under the command of Joseph O'Kean (or O'Cain), bound for the northwest coast of America. The vessel, chartered by the Russian-American Company to supply their American posts, sailed first to Japan, on to Kamchatka, and finally, in August 1807, for the Aleutian Islands.

The following month the ship was wrecked on Sanak Island, where the survivors were able to sustain themselves for two months on shellfish, berries, and some food salvaged from the ship's cargo. They were finally discovered by a party from Unalaska, who assisted them to repair and fit out their longboat for the stormy 350-mile voyage to Kodiak.

After brief stops at Unga and possibly Sutwik islands, the frail craft reached Kodiak on December 27. After three weeks for rest and refitting the 22-foot boat, the crew set out once more for Sanak to continue salvage operations. Tracking around the northern end of Kodiak Island, the party encountered foul weather, causing the boat to spring a leak and necessitating a run for the uninhabited coast.

The greater part of the crew, including Campbell, then set off on foot for the settlement of Karluk. The Scotsman's sealskin boots became soaked with water and soon his feet were completely frozen. The party tried to go on, but finally after many hardships had to return to their starting place. A second group then set out for Karluk and this time was successful in sending some three-holed baidarkas to pick up the survivors. Campbell and the others spent three weeks at Karluk, during which time his hands and feet "began to mortify." Finally, on March 23, after a long journey in an open skin boat, the ragged party reached Kodiak.

It happened that a Dr. Mordgorst (Campbell calls him Nordgoorst), surgeon of the Russian ship *Neva*, was spending the winter at the settlement. It is his account of the case that is presented below.

After his surgery Campbell remained at Kodiak until December 1808, whiling away his time teaching English to some Native children. He was finally able to take passage to Hawaii, where he remained over a year recuperating from his ordeal. He finally reached Scotland in April 1812, where for the remainder of his life he earned a scant living playing the barrel-organ and later the violin. He never walked again, largely because his amputations had been performed below the ankle and the wounds had never healed, possibly because of osteomyelitis.

"This Englishman sailed from Kodiak in the winter time, in the ship's cutter, for the island of Sannack. On their passage a storm came on, in which the boat was wrecked. The crew saved their lives on shore; but this man had both his feet frozen, and not having stripped off his clothes for twenty-seven days, he was not aware of the extent of his calamity, and did not apprehend the destruction of his feet. . . .

"In the first place I had his feet cleaned and dried; they were both in a state of mortification (gangrena sicca.) The mortified parts having separated from the sound to a distance of a finger's breadth, where either amputation might take place or a cure be performed, as the patient himself hoped. I dressed the mortified, or frostbitten parts with oil of turpentine, and the unaffected parts with olive oil, and continued these applications for about five days, after which I used charcoal, gas, and other chimical applications; but as there appeared no chance of saving his feet, I began to consider that there was no resourse left but amputation. That the patient might not be alarmed, I talked over the matter with him as is usual in such cases, and endeavoured to persuade him to submit to the operation, as the only means of effecting a cure. But at first I was not successful, and could not get him to agree to it. I was therefore obliged to continue my former mode of treatment. At the end of three days, however, he gave his consent, and I fixed a time for the operations, which I performed satisfactorily. On the third day after the operation, the wound appeared to be in a good state, and I continued to dress it daily as it required.

"The other foot remained to undergo a similar operation. I suffered three weeks to elapse, when it also took place. The wounds are now in a good state, and evidently healing up.

"It is not in my power to complete the cure, being obliged to return to Russia; but I have left the directions with the assistant surgeon how to proceed in the treatment."

REFERENCE

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President's Corner

The annual meeting in Fairbanks was deficiently attended. The program was interesting and the evening functions were entertaining. Especially notable was the dinner at Dredge #8 where our three Russian physician friends were introduced and given a chance to make a few remarks. They are highly interested in learning some of our techniques, but are limited by an economy that denies them the technological wherewithal. Everyone, including Dr. John Tupper, the incoming president of the American Medical Association, was enthused over the possibility of having next year's meeting in Magadan. There are numerous logistic hurdles, but we will continue to pursue.

The council voted to join the Western Journal of Medicine family. Every member will become a subscriber automatically with the payment of dues. *Alaska Medicine* will continue in its quarterly format. I remain the editor. We need articles - so sharpen your pencils and share your interesting cases, etc.

Donald R. Rogers, M.D.
President
Alaska State Medical Association

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President-Elect's Word

Being president of the Anchorage Medical Society for 1990 has been kind of fun. I've enjoyed arranging programs, spearheading issues, being a spokesperson, and so on. Being the first female president has seemed like a pleasant detail - a minor documentation of how times have changed.

It has therefore caught me by surprise to discover that I feel some insecurity about being the first female president of the Alaska State Medical Association. Here are some of the reasons:

1. The issues are bigger, the stakes are higher, and I am still a novice. Will I have the time and energy to learn all the critical local, statewide and national issues, and the wisdom to lead you in effective action?
2. Physicians are a tough group to work with. As those who practice both an art and a science, physicians are not an easy constituency to serve. By and large, we are demanding, opinionated, and individualistic (that's the "artist" in us). At the same time, many of us prefer to observe the fray and remain impartial (that's the "scientist" in us). On top of that, many of us have no taste for "medical politics" or "social medicine" and the like. Can I withstand the inevitable criticism and discord without losing heart myself? Can I help build a spirit of enthusiasm and greater unity?
3. In contrast to our local society, ASMA has few active women. My experience in Anchorage may be so comfortable because it is pleasantly coed. Will my "minority" status in ASMA leadership keep me feeling lonely and defensive? ASMA needs a relaxed and confident leader.

If you can join me and the rest of ASMA's leadership in our efforts to preserve the best of an honorable and rewarding profession, please do. Despite difficulties and insecurity, I will be doing my best to be an appropriate leader for the profession of medicine in Alaska in 1991-2.

Jennifer Christian, M.D.
President-Elect, ASMA

Auxiliary News

ALASKA STATE MEDICAL ASSOCIATION AUXILIARY

The Alaska State Medical Association Auxiliary convention in Fairbanks June 7 through 10, 1990 signaled the end of my first year as President and the beginning of my second term. We have some good programs planned beginning in the fall, and I hope that more of you will take advantage of the educational programs available. I don't want to rush ahead of myself, instead let me tell you about the recent convention.

Our speakers included two psychiatrists and an ob-gyn nurse practitioner. Dr. Joseph O'Lone, a psychiatrist from Fairbanks, helped us look at relationships, including marriage, with respect to our individual personal growth. Dr. Wandal Winn, an Anchorage psychiatrist spoke to us about the family problem solving that can be accomplished through organized family meetings. He also offered suggestions for fun activities and learning objectives that can be done through this type of family gathering. Phyllis Banks, M.S., P.A. spoke to us very candidly about adolescent sexuality and why kids do or do not use contraceptives. We also talked about the kinds of questions most kids have about their sexuality as well as physical development, physiology, and why they don't talk to their parents. We discussed ways to talk to our own children about this very delicate issue. All three speakers engaged their audience in free flowing discussion and hopefully we all took something helpful home with us.

Highlights of the convention were the luncheon with our gubernatorial candidates, dinner with the Russian delegation from Magadan, and of course, all the great scientific sessions available for continuing education.

Changing step now, I am looking forward to the national convention in Chicago in just two weeks. Resolutions were discussed at our convention and votes taken in preparation for the House of Delegates meeting. I will have a complete report for you in the next issue of our newsletter.

It has been a pleasure working with ASMA, the staff, and the physicians on the board. I will continue to be available for special projects and encourage our organizations to work together for the better health of Alaskans everywhere.

Lindsay A. Hansen Tucker

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Guest Editorial

RESTRUCTURING HEALTH CARE FINANCING IN ALASKA

There is considerable breast-beating in the nation over the fact that more than 30 million people do not have health care insurance. There is, however, no national consensus as to how to rectify this deplorable state of affairs.

Because of this, states one by one are wrestling with the problem. Hawaii has universal health insurance in place. Massachusetts soon will if severe underfinancing can be corrected. Oregon, Washington, and Missouri among other states have plans for universal systems.

Alaska is considerably behind other jurisdictions. There may be as many as 50,000 persons in the state without insurance coverage and many more who are grossly underinsured. Many state legislators are concerned about the problem but grope in piecemeal fashion for solutions. A bill (HB 581) was passed in the recent session to create (yet another!) task force to devise a plan for universal coverage by June 30, 1992.

There are several demographic, institutional, and fiscal realities here which might make it easier for us to design coverage for everyone in the state. It is not so much a matter of restructuring the way medical care is given as it is a question of taking advantage of present realities and resources:

· Only 4% of Alaskans are over 65 years of age compared to 12% nationally. Care of the aged is very costly.

· Indian Health Service. There is a $100 million infusion of federal money into the state to partially fund care of Alaska Natives.

· Bountiful health care plans for government workers. Perhaps these should be not quite so rich and the savings used to fund in part care for the uninsured.

· Mental Health Lands Trust Fund. Funding is still in dispute but will likely be at least $140 million yearly. Most of this will be used for institutional and outpatient mental health care.

· Pioneer Homes. We currently spend approximately $26 million annually for this aspect of long-term care.

· Longevity Bonus Program. This $62 million program could be gradually converted to long-term care for the aged.

· Permanent Fund dividends. Uninsured individuals could be given the option of using dividends to purchase $1000 deductible group insurance, subject to underwriting criteria. They might then be pooled with government workers.

· Absence of Alaska based health insurance companies. Their interests and profitability do not have to be catered to in devising new financing mechanisms.

· Absence of state income or other general taxes. Fresh ways of paying for health care may require taxation in order to take the burden off employers and to avoid present rampant cost shifting. This does not necessarily mean that more money totally would be spent on medical and dental care than the roughly $2 billion that we currently expend annually.

These existing "assets" could be used to create an administratively simpler system of paying for health care in Alaska, incorporating one way or another existing payment programs such as Medicaid, Medicare, Champus, VA, prison care, workers' compensation, and even awards for future care in medical malpractice cases.

It behooves us all to elect public officials this year who have the desire, intelligence, and drive to rectify the many inequities, extravagances, and gaps in our health care payment systems.

Rodman Wilson, M.D.
Member, Editorial Board

NEW MEDICARE CLAIMS FILING REQUIREMENT

Recent Legislation

When Congress passed the Omnibus Budget Reconciliation Act of 1989, it included a requirement that all physicians and suppliers submit claims for Medicare beneficiaries beginning September 1, 1990. Congress believed that this would yield more accurate information with which to evaluate Medicare expenditures and other factors such as volume and intensity of services under the Medicare Volume Performance Standard (MVPS). The Standard is Congress's primary tool for managing the growth in Medicare Part B expenditures for physician services. For more information on the MVPS contact the Alaska State Medical Association.

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Editor

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VASOTEC®

(ENALAPRIL MALEATE | MSD)

VASOTEC is available in 2.5-mg, 5-mg, 10-mg, and 20-mg tablet strengths.

Contraindications: VASOTEC® (Enalapril Maleate, MSD) is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor.

Warnings: *Angioedema* Angioedema of the face, extremities, lips, tongue, glottis, and/or larynx has been reported in patients treated with ACE inhibitors, including VASOTEC In such cases, VASOTEC should be promptly discontinued and the patient carefully observed until the swelling disappears In instances where swelling has been confined to the face and lips, the condition has generally resolved without treatment, although antihistamines have been useful in relieving symptoms. Angioedema associated with laryngeal edema may be fatal **Where there is involvement of the tongue, glottis, or larynx likely to cause airway obstruction, appropriate therapy, e.g., subcutaneous epinephrine solution 1:1000 (0.3 mL to 0.5 mL), should be promptly administered.** (See ADVERSE REACTIONS.)

Hypotension: Excessive hypotension is rare in uncomplicated hypertensive patients treated with VASOTEC alone Patients with heart failure given VASOTEC commonly have some reduction in blood pressure, especially with the first dose, but discontinuation of therapy for continuing symptomatic hypotension usually is not necessary when dosing instructions are followed, caution should be observed when initiating therapy. (See DOSAGE AND ADMINISTRATION.) Patients at risk for excessive hypotension, sometimes associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death, include those with the following conditions or characteristics: heart failure, hyponatremia, high-dose diuretic therapy, recent intensive diuresis or increase in diuretic dose, renal dialysis, or severe volume and/or salt depletion of any etiology. It may be advisable to eliminate the diuretic (except in patients with heart failure), reduce the diuretic dose, or increase salt intake cautiously before initiating therapy with VASOTEC in patients at risk for excessive hypotension who are able to tolerate such adjustments. (See PRECAUTIONS, *Drug Interactions* and ADVERSE REACTIONS.) In patients at risk for excessive hypotension, therapy should be started under very close medical supervision and such patients should be followed closely for the first two weeks of treatment and whenever the dose of enalapril and/or diuretic is increased. Similar considerations may apply to patients with ischemic heart disease or cardiovascular disease in whom an excessive fall in blood pressure could result in a myocardial infarction or cerebrovascular accident If excessive hypotension occurs, the patient should be placed in the supine position and, if necessary, receive an intravenous infusion of normal saline A transient hypotensive response is not a contraindication to further doses of VASOTEC, which usually can be given without difficulty once the blood pressure has stabilized If symptomatic hypotension develops, a dose reduction or discontinuation of VASOTEC or concomitant diuretic may be necessary

Neutropenia/Agranulocytosis: Another ACE inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression, rarely in uncomplicated patients but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease. Available data from clinical trials of enalapril are insufficient to show that enalapril does not cause agranulocytosis at similar rates. Foreign marketing experience has revealed several cases of neutropenia or agranulocytosis in which a causal relationship to enalapril cannot be excluded Periodic monitoring of white blood cell counts in patients with collagen vascular disease and renal disease should be considered

Precautions: *General: Impaired Renal Function.* As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including VASOTEC, may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death.

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine were observed in 20% of patients These increases were almost always reversible upon discontinuation of enalapril and/or diuretic therapy In such patients, renal function should be monitored during the first few weeks of therapy

Some patients with hypertension or heart failure with no apparent preexisting renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when VASOTEC has been given concomitantly with a diuretic. This is more likely to occur in patients with preexisting renal impairment. Dosage reduction and/or discontinuation of the diuretic and/or VASOTEC may be required

Evaluation of patients with hypertension or heart failure should always include assessment of renal function. (See DOSAGE AND ADMINISTRATION.)

Hyperkalemia Elevated serum potassium (>5.7 mEq/L) was observed in approximately 1% of hypertensive patients in clinical trials. In most cases these were isolated values which resolved despite continued therapy. Hyperkalemia was a cause of discontinuation of therapy in 0.28% of hypertensive patients In clinical trials in heart failure, hyperkalemia was observed in 3.8% of patients, but was not a cause for discontinuation

Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with VASOTEC. (See *Drug Interactions*.)

Surgery/Anesthesia In patients undergoing major surgery or during anesthesia with agents that produce hypotension, enalapril may block angiotensin II formation secondary to compensatory renin release If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion.

Information for Patients:

Angioedema Angioedema, including laryngeal edema, may occur especially following the first dose of enalapril. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to take no more drug until they have consulted with the prescribing physician.

Hypotension Patients should be cautioned to report lightheadedness, especially during the first few days of therapy If actual syncope occurs, the patients should be told to discontinue the drug until they have consulted with the prescribing physician.

All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduction in fluid volume Other causes of volume depletion such as vomiting or diarrhea may also lead to a fall in blood pressure, patients should be advised to consult with the physician.

Hyperkalemia Patients should be told not to use salt substitutes containing potassium without consulting their physician.

Neutropenia Patients should be told to report promptly any indication of infection (e.g., sore throat, fever) which may be a sign of neutropenia

NOTE: As with many other drugs, certain advice to patients being treated with enalapril is warranted This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects.

Drug Interactions.

Hypotension. Patients on Diuretic Therapy: Patients on diuretics and especially those in whom diuretic therapy was recently instituted may occasionally experience an excessive reduction of blood pressure after initiation of therapy with enalapril. The possibility of hypotensive effects with enalapril can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with enalapril. If it is necessary to continue the diuretic, provide close medical supervision after the initial dose for at least two hours and until blood pressure has stabilized for at least an additional hour (See WARNINGS and DOSAGE AND ADMINISTRATION.)

Agents Causing Renin Release The antihypertensive effect of VASOTEC is augmented by antihypertensive agents that cause renin release (e.g., diuretics).

Other Cardiovascular Agents: VASOTEC has been used concomitantly with beta-adrenergic-blocking agents, methyldopa, nitrates, calcium-blocking agents, hydralazine, prazosin, and digoxin without evidence of clinically significant adverse interactions

Agents Increasing Serum Potassium: VASOTEC attenuates potassium loss caused by thiazide-type diuretics Potassium-sparing diuretics (e.g., spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium. Therefore, if concomitant use of these agents is indicated because of demonstrated hypokalemia, they should be used with caution and with frequent monitoring of serum potassium. Potassium-sparing agents should generally not be used in patients with heart failure receiving VASOTEC

Lithium: Lithium toxicity has been reported in patients receiving lithium concomitantly with drugs which cause elimination of sodium, including ACE inhibitors. A few cases of lithium toxicity have been reported in patients receiving concomitant VASOTEC and lithium and were reversible upon discontinuation of both drugs. It is recommended that serum lithium levels be monitored frequently if enalapril is administered concomitantly with lithium.

Pregnancy – Category C: There was no fetotoxicity or teratogenicity in rats treated with up to 200 mg/kg/day of enalapril (333 times the maximum human dose). Fetotoxicity, expressed as a decrease in average fetal weight, occurred in rats given 1200 mg/kg/day of enalapril but did not occur when these animals were supplemented with saline Enalapril was not teratogenic in rabbits However, maternal and fetal toxicity occurred in some rabbits at doses of 1 mg/kg/day or more. Saline supplementation prevented the maternal and fetal toxicity seen at doses of 3 and 10 mg/kg/day, but not at 30 mg/kg/day (50 times the maximum human dose)

Radioactivity was found to cross the placenta following administration of labeled enalapril to pregnant hamsters.

There are no adequate and well-controlled studies of enalapril in pregnant women. However, data are available that show enalapril crosses the human placenta Because the risk of fetal toxicity with the use of ACE inhibitors has not been clearly defined, VASOTEC® (Enalapril Maleate, MSD) should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Postmarketing experience with all ACE inhibitors thus far suggests the following with regard to pregnancy outcome. Inadvertent exposure limited to the first trimester of pregnancy has not been reported to affect fetal outcome adversely Fetal exposure during the second and third trimesters of pregnancy has been associated with fetal and neonatal morbidity and mortality

When ACE inhibitors are used during the later stages of pregnancy, there have been reports of hypotension and decreased renal perfusion in the newborn. Oligohydramnios in the mother has also been reported, presumably representing decreased renal function in the fetus. Infants exposed *in utero* to ACE inhibitors should be closely observed for hypotension, oliguria, and hyperkalemia If oliguria occurs, attention should be directed toward support of blood pressure and renal perfusion with the administration of fluids and pressors as appropriate Problems associated with prematurity such as patent ductus arteriosus have occurred in association with maternal use of ACE inhibitors, but it is not clear whether they are related to ACE inhibition, maternal hypertension, or the underlying prematurity

Nursing Mothers. Milk in lactating rats contains radioactivity following administration of ^{14}C enalapril maleate. It is not known whether this drug is secreted in human milk. Because many drugs are secreted in human milk, caution should be exercised when VASOTEC is given to a nursing mother.

Pediatric Use Safety and effectiveness in children have not been established

Adverse Reactions: VASOTEC has been evaluated for safety in more than 10,000 patients, including over 1000 patients treated for one year or more VASOTEC has been found to be generally well tolerated in controlled clinical trials involving 2987 patients.

HYPERTENSION: The most frequent clinical adverse experiences in controlled trials were: headache (5.2%), dizziness (4.3%), and fatigue (3%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in controlled clinical trials were: diarrhea (1.4%), nausea (1.4%), rash (1.4%), cough (1.3%), orthostatic effects (1.2%), and asthenia (1.1%).

HEART FAILURE The most frequent clinical adverse experiences in both controlled and uncontrolled trials were: dizziness (7.9%), hypotension (6.7%), orthostatic effects (2.2%), syncope (2.2%), cough (2.2%), chest pain (2.1%), and diarrhea (2.1%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in both controlled and uncontrolled clinical trials were: fatigue (1.8%), headache (1.8%), abdominal pain (1.6%), asthenia (1.6%), orthostatic hypotension (1.6%), vertigo (1.6%), angina pectoris (1.5%), nausea (1.3%), vomiting (1.3%), bronchitis (1.3%), dyspnea (1.3%), urinary tract infection (1.3%), rash (1.3%), and myocardial infarction (1.2%).

Other serious clinical adverse experiences occurring since the drug was marketed or adverse experiences occurring in 0.5% to 1% of patients with hypertension or heart failure in clinical trials in order of decreasing severity within each category

Cardiovascular Cardiac arrest, myocardial infarction or cerebrovascular accident, possibly secondary to excessive hypotension in high-risk patients (see WARNINGS, *Hypotension*), pulmonary embolism and infarction, pulmonary edema; rhythm disturbances, atrial fibrillation; palpitation.

Digestive: Ileus, pancreatitis, hepatitis (hepatocellular or cholestatic jaundice), melena, anorexia, dyspepsia, constipation, glossitis, stomatitis, dry mouth

Musculoskeletal: Muscle cramps

Nervous/Psychiatric: Depression, confusion, ataxia, somnolence, insomnia, nervousness, paresthesia.

Urogenital: Renal failure, oliguria, renal dysfunction (see PRECAUTIONS and DOSAGE AND ADMINISTRATION).

Respiratory Bronchospasm, rhinorrhea, sore throat and hoarseness, asthma, upper respiratory infection.

Skin. Exfoliative dermatitis, toxic epidermal necrolysis, Stevens-Johnson syndrome, herpes zoster, erythema multiforme, urticaria, pruritus, alopecia, flushing, hyperhidrosis

Special Senses. Blurred vision, taste alteration, anosmia, tinnitus, conjunctivitis, dry eyes, tearing

A symptom complex has been reported which may include a positive ANA, an elevated erythrocyte sedimentation rate, arthralgias/arthritis, myalgias, fever, serositis, vasculitis, leukocytosis, eosinophilia, photosensitivity, rash, and other dermatologic manifestations

Angioedema: Angioedema has been reported in patients receiving VASOTEC (0.2%). Angioedema associated with laryngeal edema may be fatal If angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with VASOTEC should be discontinued and appropriate therapy instituted immediately (See WARNINGS.)

Hypotension: In the hypertensive patients, hypotension occurred in 0.9% and syncope occurred in 0.5% of patients following the initial dose or during extended therapy Hypotension or syncope was a cause for discontinuation of therapy in 0.1% of hypertensive patients. In heart failure patients, hypotension occurred in 6.7% and syncope occurred in 2.2% of patients. Hypotension or syncope was a cause for discontinuation of therapy in 1.9% of patients with heart failure (See WARNINGS.)

Clinical Laboratory Test Findings

Serum Electrolytes: Hyperkalemia (see PRECAUTIONS), hyponatremia

Creatinine, Blood Urea Nitrogen. In controlled clinical trials, minor increases in blood urea nitrogen and serum creatinine, reversible upon discontinuation of therapy, were observed in about 0.2% of patients with essential hypertension treated with VASOTEC alone Increases are more likely to occur in patients receiving concomitant diuretics or in patients with renal artery stenosis. (See PRECAUTIONS.) In patients with heart failure who were also receiving diuretics with or without digitalis, increases in blood urea nitrogen or serum creatinine, usually reversible upon discontinuation of VASOTEC and/or other concomitant diuretic therapy, were observed in about 11% of patients Increases in blood urea nitrogen or creatinine were a cause for discontinuation in 1.2% of patients.

Hemoglobin and Hematocrit. Small decreases in hemoglobin and hematocrit (mean decreases of approximately 0.3 g% and 1.0 vol %, respectively) occur frequently in either hypertension or heart failure patients treated with VASOTEC but are rarely of clinical importance unless another cause of anemia coexists. In clinical trials, less than 0.1% of patients discontinued therapy due to anemia

Other (Causal Relationship Unknown): In marketing experience, rare cases of neutropenia, thrombocytopenia, and bone marrow depression have been reported. A few cases of hemolysis have been reported in patients with G6PD deficiency

Liver Function Tests. Elevations of liver enzymes and/or serum bilirubin have occurred.

Dosage and Administration: *Hypertension.* In patients who are currently being treated with a diuretic, symptomatic hypotension occasionally may occur following the initial dose of VASOTEC. The diuretic should, if possible, be discontinued for two to three days before beginning therapy with VASOTEC to reduce the likelihood of hypotension. (See WARNINGS.) If the patient's blood pressure is not controlled with VASOTEC alone, diuretic therapy may be resumed.

If the diuretic cannot be discontinued, an initial dose of 2.5 mg should be used under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions*.)

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The recommended initial dose in patients not on diuretics is 5 mg once a day. Dosage should be adjusted according to blood pressure response The usual dosage range is 10 to 40 mg per day administered in a single dose or in two divided doses In some patients treated once daily, the antihypertensive effect may diminish toward the end of the dosing interval. In such patients, an increase in dosage or twice-daily administration should be considered. If blood pressure is not controlled with VASOTEC alone, a diuretic may be added.

Concomitant administration of VASOTEC with potassium supplements, potassium salt substitutes, or potassium-sparing diuretics may lead to increases of serum potassium (see PRECAUTIONS).

Dosage Adjustment in Hypertensive Patients with Renal Impairment The usual dose of enalapril is recommended for patients with a creatinine clearance > 30 mL/min (serum creatinine of up to approximately 3 mg/dL). For patients with creatinine clearance ≤ 30 mL/min (serum creatinine ≥ 3 mg/dL), the first dose is 2.5 mg once daily The dosage may be titrated upward until blood pressure is controlled or to a maximum of 40 mg daily

Heart Failure: VASOTEC is indicated as adjunctive therapy with diuretics and digitalis. The recommended starting dose is 2.5 mg once or twice daily After the initial dose of VASOTEC, the patient should be observed under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions*.) If possible, the dose of the diuretic should be reduced, which may diminish the likelihood of hypotension. The appearance of hypotension after the initial dose of VASOTEC does not preclude subsequent careful dose titration with the drug, following effective management of the hypotension. The usual therapeutic dosing range for the treatment of heart failure is 5 to 20 mg daily given in two divided doses. The maximum daily dose is 40 mg Once-daily dosing has been effective in a controlled study, but nearly all patients in this study were given 40 mg, the maximum recommended daily dose, and there has been much more experience with twice-daily dosing. In addition, in a placebo-controlled study which demonstrated reduced mortality in patients with severe heart failure (NYHA Class IV), patients were treated with 2.5 to 40 mg per day of VASOTEC, almost always administered in two divided doses. (See CLINICAL PHARMACOLOGY, *Pharmacodynamics and Clinical Effects*.) Dosage may be adjusted depending upon clinical or hemodynamic response. (See WARNINGS.)

Dosage Adjustment in Patients with Heart Failure and Renal Impairment or Hyponatremia In patients with heart failure who have hyponatremia (serum sodium < 130 mEq/L) or with serum creatinine >1.6 mg/dL, therapy should be initiated at 2.5 mg daily under close medical supervision. (See DOSAGE AND ADMINISTRATION, *Heart Failure*, WARNINGS, and PRECAUTIONS, *Drug Interactions*.) The dose may be increased to 2.5 mg b.i.d., then 5 mg b.i.d. and higher as needed, usually at intervals of four days or more, if at the time of dosage adjustment there is not excessive hypotension or significant deterioration of renal function. The maximum daily dose is 40 mg

For more detailed information, consult your MSD Representative or see Prescribing Information, Merck Sharp & Dohme, Division of Merck & Co., INC., West Point, PA 19486.

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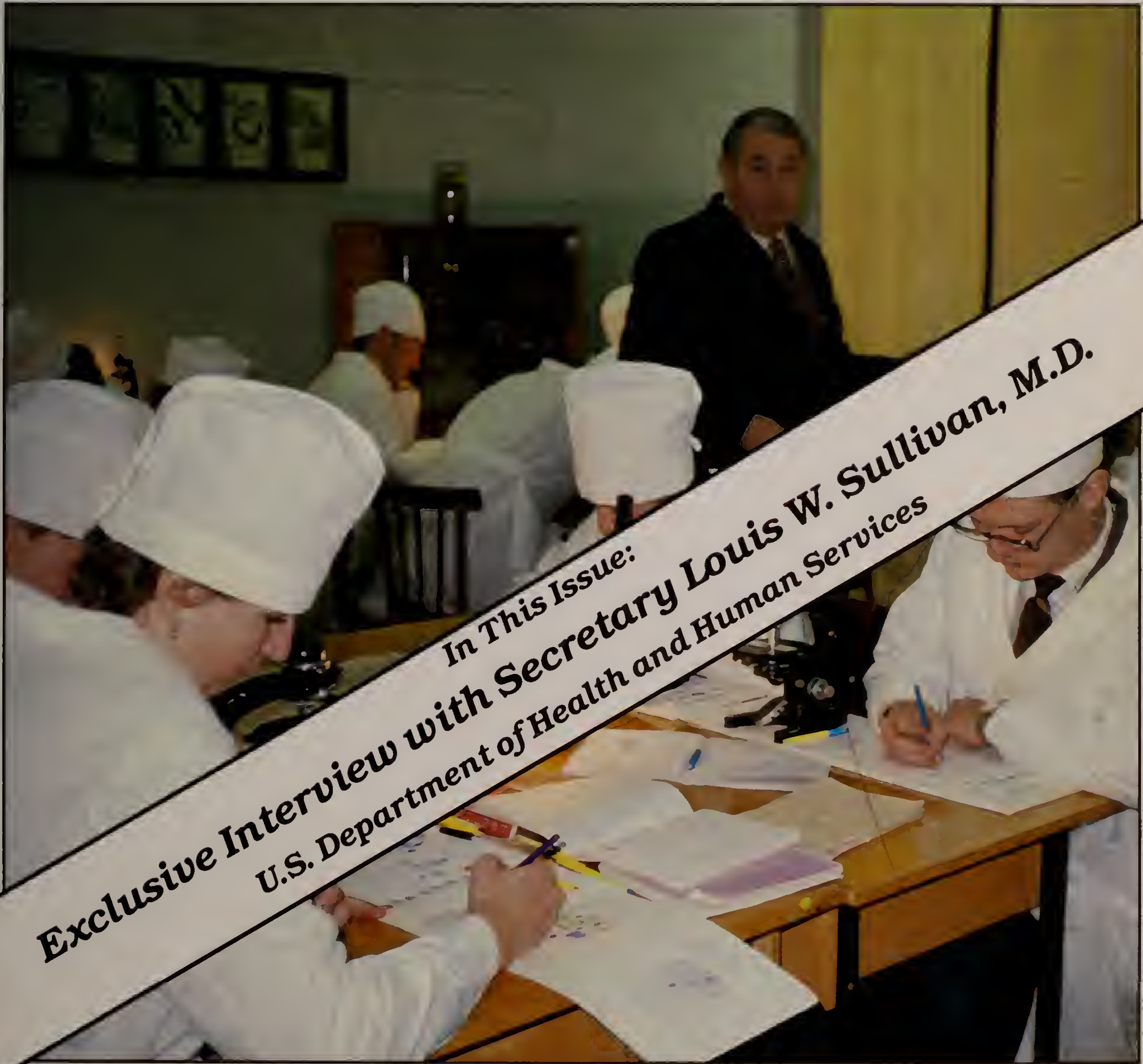

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References

1. USP DI Update, September/October 1988, p 120.
2. Br J Clin Pharmacol 1985;20:710-713.
3. Data on file, Lilly Research Laboratories.
4. Scand J Gastroenterol 1987;22(suppl 136):61-70.
5. Am J Gastroenterol 1989;84:769-774.

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Brief Summary. Consult the package literature for complete information.

Indications and Usage: 1. *Active duodenal ulcer*—for up to eight weeks of treatment. Most patients heal within four weeks.

2. *Maintenance therapy*—for healed duodenal ulcer patients at a reduced dosage of 150 mg h.s. The consequences of therapy with Axid for longer than one year are not known.

Contraindication: Known hypersensitivity to the drug. Use with caution in patients with hypersensitivity to other H_2-receptor antagonists.

Precautions: *General*—1. Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy.

2. Dosage should be reduced in patients with moderate to severe renal insufficiency.

3. In patients with normal renal function and uncomplicated hepatic dysfunction, the disposition of nizatidine is similar to that in normal subjects.

Laboratory Tests—False-positive tests for urobilinogen with Multistix® may occur during therapy.

Drug Interactions—No interactions have been observed with theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin. Axid does not inhibit the cytochrome P-450 enzyme system; therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur. In patients given very high doses (3,900 mg) of aspirin daily, increased serum salicylate levels were seen when nizatidine, 150 mg b.i.d., was administered concurrently.

Carcinogenesis, Mutagenesis, Impairment of Fertility—A two-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a two-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups. The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

Axid® (nizatidine, Lilly)

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a two-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy—Teratogenic Effects—Pregnancy Category C—Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in one fetus, and at 50 mg/kg, it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in one fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers—Studies in lactating women have shown that 0.1% of an oral dose is secreted in human milk in proportion to plasma concentrations. Because of growth depression in pups reared by treated lactating rats, a decision should be made whether to discontinue nursing or the drug, taking into account the importance of the drug to the mother.

Pediatric Use—Safety and effectiveness in children have not been established.

Use in Elderly Patients—Healing rates in elderly patients were similar to those in younger age groups as were the rates of adverse events and laboratory test abnormalities. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Clinical trials of varying durations included almost 5,000 patients. Among the more common adverse events in domestic placebo-controlled trials of over 1,900 nizatidine patients and over 1,300 on placebo, sweating (1% vs 0.2%), urticaria (0.5% vs <0.01%), and somnolence (2.4% vs 1.3%) were significantly more common with nizatidine. It was not possible to determine whether a variety of less common events was due to the drug.

Axid® (nizatidine, Lilly)

Hepatic—Hepatocellular injury (elevated liver enzyme tests or alkaline phosphatase) possibly or probably related to nizatidine occurred in some patients. In some cases, there was marked elevation (>500 IU/L) in SGOT or SGPT and, in a single instance, SGPT was >2,000 IU/L. The incidence of elevated liver enzymes overall and elevations of up to three times the upper limit of normal, however, did not significantly differ from that in placebo patients. Hepatitis and jaundice have been reported. All abnormalities were reversible after discontinuation of Axid.

Cardiovascular—In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in two individuals administered Axid and in three untreated subjects.

CNS—Rare cases of reversible mental confusion have been reported.

Endocrine—Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to nizatidine. Impotence and decreased libido were reported with equal frequency by patients on nizatidine and those on placebo. Gynecomastia has been reported rarely.

Hematologic—Fatal thrombocytopenia was reported in a patient treated with nizatidine and another H_2-receptor antagonist. This patient had previously experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental—Sweating and urticaria were reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity—As with other H_2-receptor antagonists, rare cases of anaphylaxis following nizatidine administration have been reported. Because cross-sensitivity among this class has been observed, H_2-receptor antagonists should not be administered to those with a history of hypersensitivity to these agents. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other—Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine have been reported.

Overdosage: Overdoses of Axid have been reported rarely. If overdosage occurs, activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. Renal dialysis for four to six hours increased plasma clearance by approximately 84%.

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Trends and Patterns of Postneonatal Mortality in Alaska 1977 through 1984

Harry Harrison Jr., M.D. (1).

ABSTRACT

Several determinants of Alaska postneonatal mortality rate were examined to determine whether very low birth weight (<1500 g) and low birth weight (1500-2499 g) surviving neonates contributed to the lack of improvement in postneonatal mortality rate. For an eight year period (1977 through 1984), all infant deaths (n= 1020) of the 82,301 resident births were reviewed through vital statistics data. The infant's place of birth, ethnic group, birth weight and age at death were available. Ethnic and rural-specific postneonatal mortality rate (PNMR) significantly improved, but there was no change in birth weight distribution or birth weight-specific postneonatal mortality rate. However, surviving very low birth weight infant proportion of postneonatal mortality has increased from 5% to 8.2% and low birth weight proportion has remained unchanged (15.6%). These proportions were not offset by the decline in normal birth weight (>2500 g) postneonatal mortality rate. This trend suggests that infants born weighing <2500 g have slowed the decline of the postneonatal mortality rate.

INTRODUCTION

Alaska has one of the highest infant mortality rates in the Western United States and to a large extent, it is the result of a high postneonatal mortality rate (PNMR)(7). The high PNMR and relative lack of decline has also been experienced by the U.S as a whole when compared to other industrialized nations(12).

Reductions in support of social programs and national economic downturn have been implicated as possible reasons for lack of improvement in the PNMR (11). Postneonatal mortality has long been used as an indicator of infant health and is closely associated with the environmental conditions in which the infant must live (16). It is not surprising that adult health is closely linked to infant health, since high-risk environments should also have adverse effects on this population (1).

There has been controversy which indicates that neonatal intensive care survivors have contributed an inordinate pressure on PNMR (5,6,10). The potential consequences of increased survival of the high-risk infant during the neonatal period may be the increased postneonatal mortality rate. This can be explained by two possibilities. One, is that sick neonates may survive past 27 days in an intensive care setting, but succumb later and be classified as postneonatal mortality. Second, survived prematures or low birth weight neonates (NICU graduates) may have greater risk of postneonatal mortality.

This study was undertaken to determine trend and patterns of Alaska postneonatal mortality. It will take a close look at a number of specific background reasons such as birth weight-specific, ethnic-specific, and rural versus urban pattern of postneonatal mortality in Alaska to determine any potential means to improve the postneonatal mortality rate.

METHOD

The study population consisted of live-born infants in Alaska from 1977 through 1984 who died in infancy (prior to the first birthday). Neonatal mortality included infants who died ≤27 days of age and postneonatal mortality included infants who died between 28 days and one year. Rates for postneonatal mortality were obtained from the National Center for Health Statistics (NCHS) monthly vital statistics report and-Wegman's Annual Infant Mortality Summary (14), except those used to determine ethnic and geographic origin of the Alaskan infant. These rates were obtained from the State's Section for Vital Statistics.

Information was available on ethnic group, age at death, place of residence, mother's place of residence and birth weight. Residence was defined as borough of maternal residence at the time of birth. All Native data refers to ethnic designations of Native, Black and Other. These ethnic groups were combined because of the small number of births and deaths in the Black and

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Other categories. The ethnicity of each infant was determined by the mother's ethnic designation. Nonresident deaths were excluded since some deaths could not be linked to a birth certificate.

Rural, urban, ethnic and birth weight-specific mortality rates were compared using Chi-square test. A p-value of <0.05 was considered significant at the 95% confidence interval (CI). A relative risk was generated when comparing ethnic and rural postneonatal mortality rate. The relative risk ratios compared the probability of mortality to an exposed versus a nonexposed population (13). The contribution of confounding factors to postneonatal mortality, such as socioeconomic status, maternal education, marital status, and seasonality, were not considered in this study.

Birth weights were distributed from <1500 g (very low birth weight, VLBW), 1500-2499 g (low birth weight, LBW), and >2500 g (normal birth weight, NBW). Birth weight distribution was defined as the number of births occurring in a specific birth weight category divided by the total number of live births and expressed as a rate per thousand. Birth weight-specific infant mortality rate was defined as the number of deaths per 1000 live-born infants in that category and birth weight-specific PNMR was defined as number of deaths per 1000 surviving neonates in that category. Gestational age was not used in this study because it is far less accurate than birth weight.

Postneonatal mortality in a small area, such as a section of the State, may vary from year to year and the numbers are often too small to discern a meaningful relationship. Taking the aggregate mortality data in less populated states (such as Alaska) may still reveal too few births and deaths for a meaningful analysis. However, combining data over an extended period will diminish the yearly aberrations and yield a study population large enough to apply statistical analysis (9). In addition to aggregate data, trend analysis of PNMR may also help to provide meaningful information for health care planning, implementation and program evaluation (5).

Alaska is geographically divided into census areas by the Section of Vital Statistics, Division of Public Health. The census area has the responsibility of collecting and reporting vital records to the agency. This study used rural and urban designations as defined by the U.S. Bureau of the Census used for 1980 census (3). Thus, the urban area is census area number 13 (Anchorage borough) and the remainder of the State is considered rural. The total study was split in two periods. Period 1 was 1977 through 1980 and period 2 was 1981 through 1984.

RESULTS

In Table 1 and graph 1, infant mortality was categorized to neonatal and postneonatal mortality. Since 1977, there was a progressive decline in NMR (p=NS), but a slight increase in PNMR (p=NS). Period 1 combined NMR was 8.5 (CI 6.2-11.2) and PNMR was 5.8 (CI 4.0-8.0). The gap in combined period 2 NMR (6.4, CI 3.7-9.3) and PNMR (6.2, CI 4.0-8.6) closed.

Resident births increased 48% (from 8400 in 1977 to 12500 in 1984), but there was no change in the birth weight distribution during this time (p=NS). Table 2 shows that period 1 birth weight distribution was similar to period 2 for all categories. There was a small increase in the VLBW category from period 1 to period 2 (p=NS). Overall, the percentage of infants weighting less than 2500 g at birth decreased: 5.5% in period 1 to 4.9% in period 2. This compares favorably with U.S. birth weight distribution (6.7% in 1984) (15). Birth weight-specific infant mortality rates for 1977 through 1984 are presented in Table 3. There was a decline in birth weight-specific mortality rate in all categories from period 1 compared to period 2. LBW infant mortality showed the largest decline (p=NS).

Table 1

		Mortality Rate*	
Year	Birth	Neonatal	Postneonatal
1977	8433	8.5(6.2-11.2)	5.7(3.4-8.4)
1978	8897	9.5(7.7-12.2)	5.8(4.0-7.9)
1979	9184	9.9(7.5-11.1)	5.5(3.2-8.2)
1980	9608	6.4(3.9-8.9)	5.9(4.7-7.3)
1981	10203	7.5(5.7-9.3)	6.1(5.5-6.8)
1982	11354	5.5(3.7-7.3)	6.2(5.4-7.0)
1983	12090	7.3(5.8-8.8)	5.8(4.0-7.9)
1984	12532	5.4(4.0-6.8)	6.6(4.5-8.6)

(95% confidence interval in parentheses)
*per 1000 live births

Graph 1

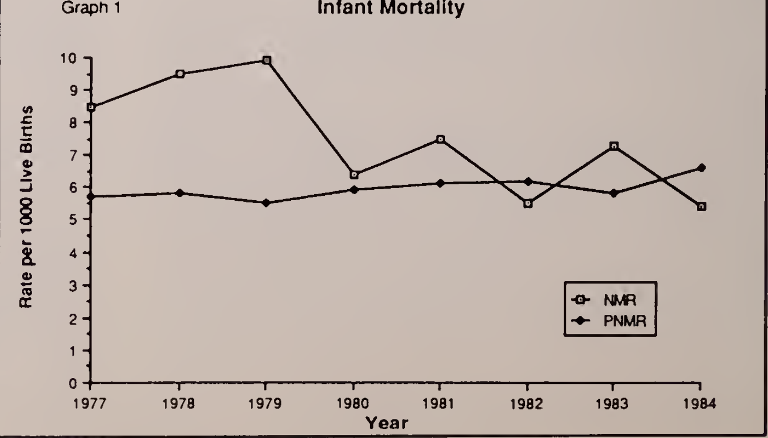


Table 2

Period	Resident Births	Birth Weight					
		<1500 g	Rate	1500-2499 g	Rate	>2500 g	Rate
1	36122	316	8.7	1681	46.5	34124	945
2	46179	410	8.9*	1862	40.3*	43908	951*

*p=NS

Table 3

Birth Weight-Specific Infant Mortality

	<1500 g				1500-2499 g				>_2500 g			
	Births	%Bths	Dths	Rate	Births	%Bths	Dths	Rate	Births	%Bths	Dths	Rate
Period 1	316	0.9	173	547	1681	4.7	88	52	34124	94.5	237	6.95
Period 2	410	0.9	173	422*	1862	4.0	75	40**	43908	95.1	274	6.24

* p=0.2
** p<0.2

Postneonatal mortality rate (per 1000 live births) increased from 5.7 in 1977 to 6.6 in 1984 (p=NS)(graph 1). However, birth weight-specific PNMR (per 1000 surviving neonates) did not change from period 1 compared to period 2 (p=NS (Table 4). Birth weight-specific PNMR for period 1 was 73 for VLBW, 21 for LBW, 5.17 for NBW and for period 2 was 77 (VLBW), 21 (LBW) and 4.24 for NBW infants. Of the 409 VLBW surviving neonates, 31 did not survive to their first birthday for a PNMR of 76 (CI 56-92). Seventy-three of the 3448 LBW survivors did not survive past the first birthday for a PNMR of 21 (CI 15-28). Surviving neonates with NBW had a PNMR of 4.6 (CI 3.2-5.8).

The ethnic-specific PNMR for non-Native and Native surviving neonates declined from 1977 through 1984 (CI non-Native, 3.5-4.8; Native, 5.3-14.4). Native PNMR significantly declined during the combined study period (p<.04). The ethnic-specific relative risk trend shows no improvement during this time indicating no shift in the ethnic-specific PNMR (1.9, CI 1.7-2.1).

Surviving neonates were further categorized to rural or urban and residence-specific PNMR was determined (Table 5). There was a decline in PNMR for both rural and urban (p<.05 and p=NS, respectively). While relative risk for rural versus urban PNMR decreased from 1.4 to 1.2 (p=NS), the overall relative risk

Table 4

Birth Weight-Specific PNMR

	<1500 g			1500-2499 g			>2500 g		
	#SN	Dths	Rate	#SN	Dths	Rate	#SN	Dths	Rate
Period 1	150	11	73	1628	35	21	34036	176	5.17
Period 2	259	20	77*	1820	38	21*	43422	186	4.24*

#SN	Number of surviving neonates
Dths	Deaths in this category
Rate	per 1000 surviving neonates
*	p=NS

in this category was 1.6 (CI 1.2-2.0). Relative risk for period 1 was 1.5 and period 2 was 1.7 (p=NS) indicating no change in relative risk. Graph 2 shows a declining trend in NBW PNMR, but no discernible change in PNMR (of surviving neonates) for VLBW and LBW infants.

DISCUSSION

For the combined study, neonatal mortality rate fell from 9.9 to 5.4, a 45% decline. By contrast, the postneonatal mortality rate increased by 11% from 5.7 to 6.6. The infant mortality rate for each birth weight category declined. However, we found no decline in PNMR for LBW infants and a small increase in the rate for VLBW babies. These findings indicate that surviving LBW and VLBW infants have slowed the decline of Alaska PNMR. The proportion of postneonatal deaths attributable to VLBW surviving neonates increased from 5% in period 1 to 8.2% in period 2 (64% increase). The increase in PNMR from period 1 to 2 can be accounted for by this group of surviving neonates. This trend has been reported in New York, Georgia and Massachusetts (2,4,18).

Since birth weight distribution did not change during the combined study period, the proportion of VLBW and LBW neonates remained unchanged. State and federally funded programs, such as Improved Pregnancy Outcome, should look to other perinatal measures for validity (8). Birth weight-specific PNMR (of surviving neonates) did not change from 1977 through 1984. Regionalization of neonatal and perinatal care (after 1980) did not change birth weight-specific PNMR when compared to pre-regionalization rate (1977-1980). This is not surprising since regionalized care did not include a component to improve postneonatal mortality rate.

Ethnic-specific PNMR of surviving neonates has declined; however, Native PNMR has shown significant improvement. Therefore, decline in the PNMR was largely attributed to improved Native PNMR. Regardless of the decline in Native PNMR, the ethnic-specific relative risk showed no change. A significant change in Native PNMR was offset by a small change in non-Native PNMR.

While rural PNMR showed a significant decline, the relative risk when compared to urban PNMR did not improve. This indicates that a small decline in urban PNMR must be matched by a much larger decline in rural PNMR to change the relative risk. The relative risk trend line indicates that the Native or rural surviving neonate remains at nearly 1.6 times the mortality risk than their non-Native or urban counterpart. Since only 50% of the non-Native births are urban, this places rural non-Native surviving neonates at the same risk as the rural Native infant. Apparently, for surviving rural neonates, PNMR is not ethnic-specific.

Neonatal regionalized care has made a major impact on decreasing the NMR in Alaska and there is evidence that perinatal (maternal-fetal) regionalization may further improve neonatal and maternal morbidity (17). The lack of improvement in PNMR may also benefit from a systematic approach to pediatric intensive care regionalization and considerable

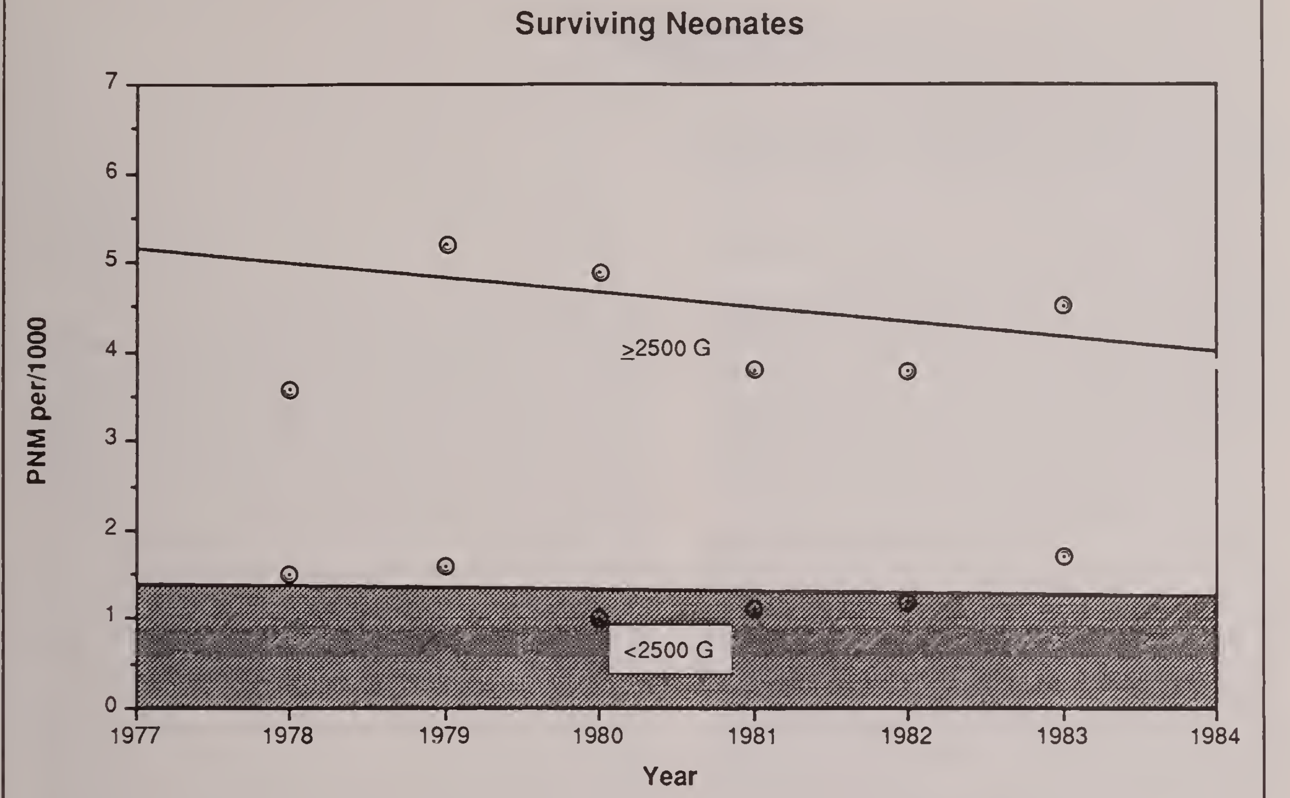
Table 5

Residence-Specific PNM Risk

Year	Surv Neo	Rural PNM	Rural Rate	Urban PNM	Urban Rate	RR	95% CI
1977	8364	37	7.9	21	5.7	1.4	0.8-2.0
1978	8812	28	5.6	16	4.2	1.3	0.7-2.0
1979	9094	42	7.9	17	4.5	1.8	1.1-2.2
1980	9545	37	6.6	18	4.6	1.5	0.9-2.1
1981	10125	28	4.7	20	4.8	1.0	0.6-1.8
1982	11283	41	6.2	14	3.0	2.1	1.3-2.9
1983	12014	56	8.0	16	3.2	2.5	1.7-3.6
1984	12460	43	5.9	26	5.0	1.2	0.7-1.9
Relative Risk						1.6	0.7-1.9

Surv Neo = Surviving neonates
PNM = Postneonatal mortality
RR = relative risk
CI = Confidence interval

Graph 2



attention to the surviving VLBW and LBW infant by primary medical caregivers. The high risk perinatal environment, surviving VLBW/LBW, and rural infant seem to carry with them the same risk factors which regionalized health care has successfully addressed for the neonate. A comprehensive family/infant program could be approached if improvement in maternal morbidity, birth weight distribution, neonatal morbidity and postneonatal mortality were considered vital at its' inception.

Further study is indicated to determine what proportion of neonates succumb while still in an intensive care setting or have a higher risk of mortality once discharged. The postneonatal mortality rate must be examined as a composite of component rates before we can appropriately direct our scarce resources.

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Advances in Anterior Cruciate Ligament Surgery

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Geri L. Grinstead, Ph.D.(2)

ABSTRACT

The anterior cruciate ligament is the most frequently torn ligament in the knee. Diagnosis of cruciate ligament tears is often a difficult task. Tests typically used in diagnosis are the anterior drawer test, Lachman's test, pivot shift test, and the flexion rotation drawer. Arthroscopy has increasingly become the standard for the diagnosis of anterior cruciate ligament tears. Management of these tears has been controversial. The diagnostic techniques are discussed and a surgical technique is described for anterior cruciate ligament reconstruction as an outpatient procedure. The middle one-third of the patellar tendon is used as a graft, harvested subcutaneously, and fixed with interference fit screws. The postoperative function of anterior cruciate ligament deficient knees has been quite satisfactory. Morbidity is decreased relative to open surgical inpatient procedures. Finally, overall cost to the patient has been significantly reduced.

Injury to the anterior cruciate ligament has been recognized for a number of years. The treatment and prognosis associated with injuries to the anterior cruciate ligament have generated considerable controversy (1). Bracing was accomplished for this injury, as early as 1850, and the first attempted repair was reported in 1900 (1). Over the years there have been a multitude of diagnostic procedures and treatment recommended. The recommendations have varied from no treatment to open surgery. The prognostic implications have ranged from the opinion that the ligament was not of importance (2) to the opinion that all ligaments should be repaired (3).

Anterior cruciate ligament tears frequently masquerade as sprains of the knee. The patient may report a sudden episode of giving way, or extreme pain in the knee. There may or may not be an audible pop or snap associated with the injury. Quite frequently the injury occurs in a non-contact sport setting. A classic example is that of the basketball player alone on the court, who stops or cuts suddenly and has the knee give way with no one else around. This clinical setting invariably is consistent with an anterior cruciate ligament tear. In the past, conservative treatment was usually recommended because the results of cruciate ligament surgery were capricious and the surgery itself was complex with significant morbidity.

CLINICAL DIAGNOSIS

The anterior cruciate ligament is the most frequently totally torn ligament in the knee and diagnosis is frequently a difficult task (4). The anterior drawer test and Lachman's test are commonly used (5). Additional tests include the pivot shift test as described by Galway (6) and the flexion rotation drawer described by Noyes (7). Opinions vary as to the accuracy and efficacy of these tests. Probably the Lachman's test is the most efficient and easy to perform. The patient is examined in the supine position. Patient relaxation is absolutely essential and difficult to achieve in the acutely injured patient. To perform this test in the right knee, the right tibia is grasped with the examiner's right hand and the right femur with the examiners left hand. The opposite hand position applies to the left knee. Maintaining the knee at 20 to 30 degrees of flexion, an anterior distraction force is applied on the tibia. This allows anterior translation of the tibia relative to the femur. A number of repetitions may be required to elicit a positive response. In general, translations of the tibia greater than 5 mm are suspect and translations greater than 1 cm are diagnostic of an anterior cruciate ligament rupture.

THE SURGERY CONTROVERSY

Recent surgical developments have made the recognition of cruciate ligament tears of more practical importance. There continues to be substantial controversy as to the proper treatment of tears of the anterior cruciate ligament, both in the acute and chronic setting. The relatively recent development of an arthroscopic approach to the reconstruction of the anterior cruciate ligament tears widens the scope of patients for whom cruciate ligament reconstruction, whether acute or chronic, may be appropriate.

---

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In his 1983 paper Johnson noted, "there continues to be controversy as to the natural history of a cruciate ligament deficient knee"(4). Gradually however, the picture seems to be clearing. It appears more likely than not that patients continue to have difficulty and subsequent re-injury to other structures within the joint in the face of a deficient anterior cruciate ligament (6). The meniscal cartilages are at risk with an anterior cruciate ligament deficient knee. Further there is substantial evidence that a medial meniscectomy may in fact increase the instability associated with an absent anterior cruciate ligament (8,9).

Because of the difficulty in initial diagnosis, there is frequently substantial time between the original injury and the ultimate recognition of the anterior cruciate ligament tear. By the time the cruciate injury is recognized, there is substantial associated intra-articular pathology (10). There is increasing evidence that the greater the elapsed time from injury to diagnosis and appropriate treatment, the greater are the intra-articular changes associated with anterior cruciate ligament disruptions (11). Three-fourths of patients with an anterior cruciate ligament injury greater than nine months prior to arthroscopic evaluation can be expected to have articular cartilage lesions(11). Conversely, fewer than 20% of patients will have articular lesions when the cruciate ligament tear occurred less than six months prior to treatment (11).

A multiplicity of surgical techniques have been advocated for anterior cruciate ligament deficient knees. In the 1970s a procedure was advocated which was alleged to increase tibial internal rotation during the active flexion phase (12). It was supposed that this would benefit the instability associated with the cruciate ligament tear. The reality is that internal rotation of the tibia produces the most unstable position for the anterior cruciate ligament tear. It is ironic that as our understanding improves, we recognize that our understanding of less than a decade ago was exactly wrong.

A variety of surgical approaches have been recommended. Both intra-articular and extra-articular procedures have been advocated. Intra-articular procedures with autogenous, allographic, and artificial materials have been advocated. All of these procedures have met with varying degrees of success or failure. The recent development of an arthroscopic procedure utilizing the middle third of the ipsilateral patellar tendon has allowed excellent results to be obtained in the outpatient setting. The ability to perform this surgery on an outpatient basis represents substantial progress both from the point of view of cost savings and from the obvious decrease in morbidity associated with the surgery. We have successfully performed the anterior cruciate ligament reconstruction by this method in over 100 consecutive patients on an outpatient basis. The results to date have been eminently satisfactory and with the decreased morbidity associated with this technique it it is now feasible to consider anterior cruciate ligament reconstruction in selected patients, as the procedure of choice for acutely injured knees.

As greater experience is gained and the technique perfected the spectrum of patients for whom primary anterior cruciate ligament reconstruction is appropriate is enlarging. Whereas in the past most patients were initially managed conservatively with bracing and operative treatment only for secondary complications, it is now feasible and practical to recommend reconstruction of the anterior cruciate ligament injury before secondary changes commence. It is our opinion that any patient under the age of 30 with a rupture of the anterior cruciate ligament sufficient to produce clinically significant pathologic instability should be offered the opportunity for primary cruciate ligament reconstruction. Those under 30 tend to lead a very active life and will, in the long term, do better with reconstruction. As for those patients over 40, relatively fewer will fit the criteria. The number of patients expected to be candidates for cruciate ligament reconstruction above the age of 50 is relatively small. Naturally there are exceptions to the rule and this is not to say that managing cruciate ligament tears non-operatively is contraindicated. With the skeletally immature individual, arthroscopy with suture of the menisci, if necessary, and conservative management of the cruciate ligament until closure of the physes, is our currently recommended treatment modality. Nevertheless, it is increasingly clear that knees with anterior cruciate ligament injuries must be *managed* either by operative or non-operative means. It is not acceptable to simply ignore the injury and, therefore, it is imperative that the injury be diagnosed.

In addition to the history and physical examination, which may be sufficient to allow accurate diagnosis of the anterior cruciate ligament tear, arthroscopy has become increasingly important. In athletes suspected of a cruciate ligament injury, the diagnosis is imperative or their careers will be curtailed. Whether a successful athletic career may be pursued with an anterior cruciate ligament deficient knee is still controversial. That it can be pursued with ignorance of the injury is no longer controversial. Clearly, athletes with cruciate ligament injuries must be recognized and treated, either operatively or non-operatively, if they are to have a successful athletic career. In our opinion, if there is substantial doubt as to the presence or absence of an anterior cruciate ligament tear in the acutely injured athlete, arthroscopy is presently the most appropriate diagnostic procedure. CT scans and MRI examination show increasing promise, but as yet, are not sufficiently reliable to exclude the possibility of the anterior cruciate ligament tear.

SURGICAL TECHNIQUE

The middle one-third of the patellar tendon can be harvested subcutaneously. The incisions necessary to harvest this graft are relatively small and can be placed transversely, paralleling Langer's lines and are therefore more cosmetic (Figure 1). Noyes (13) has shown that the central one-third of the patellar tendon at 14mm in width is 175% stronger than the normal anterior cruciate ligament, and the medial one-third (15 mm) is 163% stronger. The central one-third therefore represents a readily available and acceptable cruciate substitute (Figure 2). In our series of patients, we have encountered two instances of patellar fracture, post-operatively, following harvesting of the graft by this method. In addition, we have encountered one episode of fibrosis of the patellar tendon with subsequent patella infera requiring open release of the tendon to re-establish satisfactory function. This compares favorably with complications reported with other methodologies.

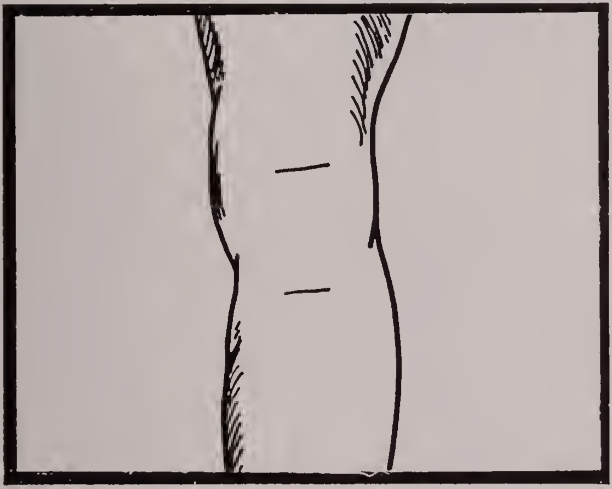

Figure 1. (a) Illustration of incisions paralleling Langer's lines.

Figure 1. (b) Demonstrates subcuticular tunneling to obtain graft.

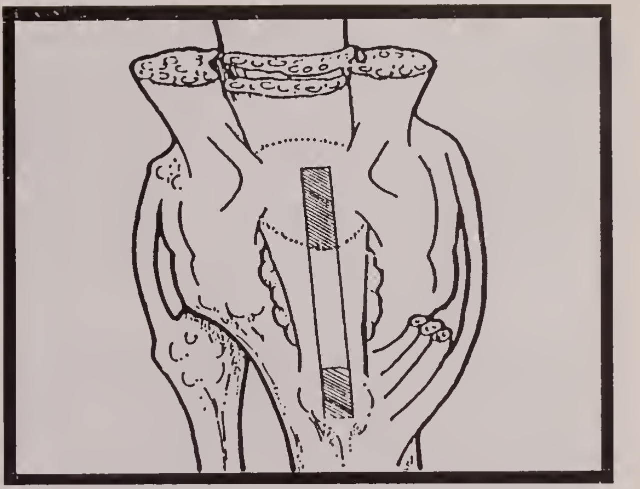

Figure 2. Diagram of the middle 1/3 of the patellar tendon to be harvested.

The key to successful anterior cruciate ligament reconstruction is accurate anatomical isometric placement of the graft. An integral part of the procedure is a notchplasty to allow sufficient room in the intercondylar notch for the new ligament substitute. In addition, the notchplasty allows adequate visualization so as to be able to place the ligament isometrically. The tibial bone tunnel is created through a small anteromedial incision. A tibial guide wire is first placed through this incision and a cannulated reamer is then used to place the 11 mm tibial tunnel accurately (Figure 3).

Figure 3. Drilling of the tibial bone tunnel using a guide pin and cannulated reamer.

Through the tibial tunnel, drilled at 11 mm, a 10 mm cannulated reamer is used to place the femoral tunnel. This obviates the need for posterolateral incisions and creates tunnels which are precisely parallel. The graft is then manipulated into position under arthroscopic control by means of a suture placed through one end of the graft. This suture is passed with a Beath pin through a separate puncture wound laterally (Figure 4).

Figure 4. Passing of graft with Beath pin.

The femoral component of the graft is then fixed in the femoral tunnel by retrograde insertion of a headless cannulated screw which functions in an interference fashion (Figure 5). This allows excellent fixation of the femoral component with minimal morbidity and the absence of posterolateral incisions. The excellent fixation in turn allows early motion which is an integral part of the reconstruction. Once the femoral portion is fixed, the tibial portion is then fixed with an interference fit screw with appropriate tension placed on the graft. At the conclusion of the arthroscopic procedure, the anterior tibial translation should be 1 to 3 mm. The graft then goes on to bony union with the femoral and tibial tunnels and the patellar tendon fibers already in continuity with the patellar tendon and tibial tubercle attachments remain in continuity, thereby providing excellent substitution for the injured cruciate ligament.

Figure 5. (a) Cannulated interference fit screw placed in femoral tunnel.

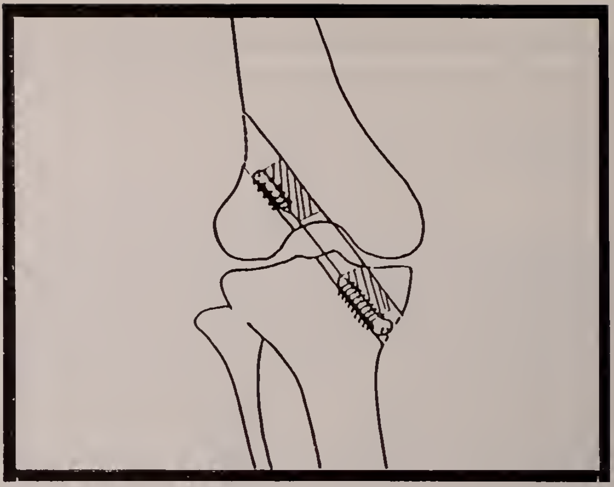

Figure 5. (b) Illustration showing screw and graft placement.

DISCUSSION

Since there are no capsular incisions using this technique, early motion is allowed. In addition, the methodology of fixation is so secure, that there is little danger of loss of fixation of the graft with early motion. Early motion minimizes the possibility of extensive adhesions and subsequent loss of motion. Finally, the ultimate viability of the graft is not compromised by early motion. The rehabilitation post-anterior cruciate reconstruction is of considerable importance and must be carried out meticulously. It is an integral part of the surgical reconstruction. This technique has allowed us to routinely perform anterior cruciate ligament reconstruction on an outpatient basis. The reconstruction has allowed many athletes to return to their chosen sport without the continuing threat of recurrent injury. As can be seen in Figure 6 the results are cosmetic. Clearly the cruciate ligament cannot be reconstructed if the diagnosis is not made. Before an athlete's knee, or for that matter, anyone's knee, is treated as a sprain, it must be clearly established that the injury does not represent a tear of the anterior cruciate ligament. The clinical examination may be sufficient to allow accurate determination of the presence or absence of a cruciate ligament injury. In those instances in which there is doubt, radiologic procedures such as the MRI or the CT scan may be helpful. At present the arthroscope represents the standard in terms of diagnostic accuracy of cruciate ligament injuries.

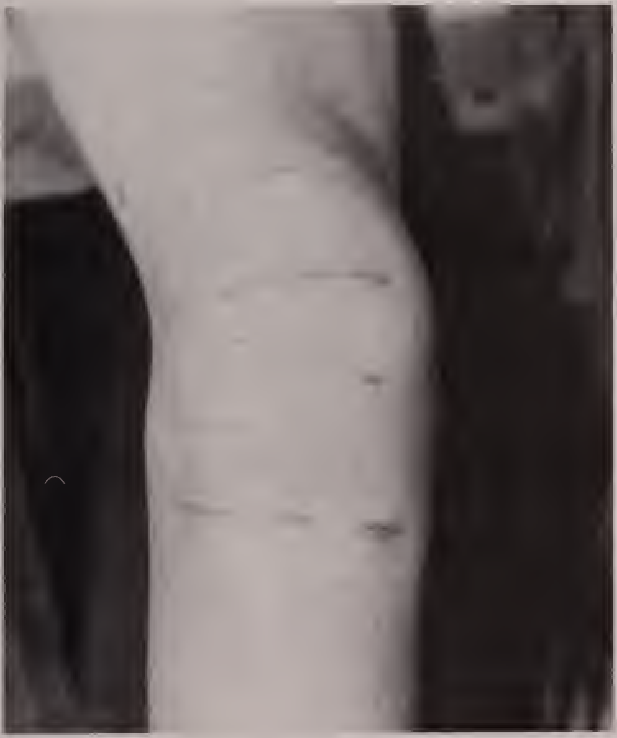

Figure 6. (a) Healed incisions at 6 weeks post ACL reconstruction.

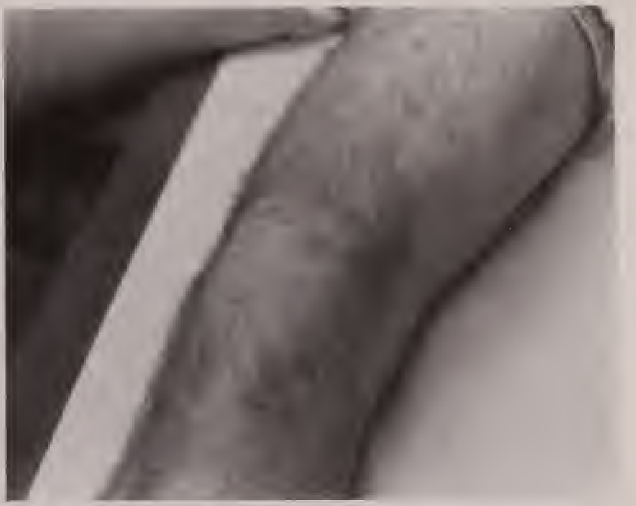

Figure 6. (b) Healed incisions at 6 months post ACL reconstruction.

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Pain Management Case Reports

Leon H. Chandler, Jr., M.D.

The Alaska Surgery Center Pain Unit began approximately two years ago in the Alaska Surgery Center, 4001 Laurel Street, Anchorage, AK 99508. Heads of the Pain Unit are Leon H. Chandler, Jr., M.D., and Michael C. Norman, M.D., who are anesthesiologists in the local community. We have addressed chronic and acute pain problems and have enjoyed a positive response from the local community and physicians. This is a summary of three recent cases that might be of interest to all physicians in the state.

CASE 1.

A 36-year-old white female was referred to the pain unit on 6/27/90 with a diagnosis of fractured rib on the left at T-9 and a sprained knee. She was referred by a local orthopedic physician for evaluation of difficulty with respiration and severe pain from the fractured rib. A block of the affected rib was done at the posterior axillary line with 5 cc of 0.75% Marcaine. She had total pain relief, was sent home, to return the following day for a pleural catheter. The patient returned on 6/28/90 and Dr. Chandler placed a pleural catheter in the left interpleural space at the T-3 area in the anterior axillary line with the aid of local xylocaine. A test dose of 5 cc of 0.50% Marcaine with epinephrine was given. After evaluation, 15 cc more of 0.50% Marcaine with epinephrine was given with resulting total pain relief. The patient was able to breathe normally. A PCA pump was then attached to the catheter which was anchored at the skin level with tape and antibiotic ointment placed over the site of injection. Two x-rays were taken, one at the time of catheter placement and one three hours later to check for pneumothorax. These were normal and the patient was placed on 8 cc of 0.25% Marcaine/hour through a continuous injection Analgesia CADD pump.

Over the next 12 days, the patient had the catheter and pump in place and received Marcaine throughout the day at a constant infusion rate of 8 cc per hour.

The patient tolerated this well and was followed by Dr. Chandler and Dr. Norman as well as Larry Smith, pharmacist, who assisted in the CADD pump monitoring and a home health care team of nurses. On 7/9/90 the pleural catheter was removed, a band-aid placed over the site, and the patient was released.

The patient felt that the pain relief from the block was very satisfactory, and she was able to carry on normal life while the rib underwent healing.

CASE 2

On 3/30/90 an 83-year-old white female was referred for acute pain and rash on the face and ear on the right side of 48 hours duration. The patient was evaluated by Dr. Norman who diagnosed shingles in the C-2 nerve root distribution on the right. A single stellate ganglion block on the right using a 27 gauge needle with 7 cc of 0.25% Marcaine was done. The patient tolerated the procedure well and within 5 minutes ear pain ceased and a 3 degree temperature rise in the right upper extremity occurred. She did not develop ptosis.

Several follow-up evaluations were done, the last in mid-April, 1990, showed that the patient had complete relief from the shingles syndrome with no post-herpetic symptoms noted.

CASE 3.

A 56-year-old female was referred to the Alaska Surgery Center Pain Unit in the terminal phases of breast carcinoma. She had significant pain of a burning nature in the ulnar distribution of the right hand, so she could not hold a newspaper. She had metastasis to the right hip, liver and other areas of the chest. She was on mega-doses of analgesics when she presented at the Pain Clinic, including Mepergan, Morphine, Roxanol, Motrin, Zanax, Desyrel and Haldol. In addition, on the 8th of June we started her on Morphine I.V. through a Patient Controlled Analgesia CADD pump at 5 mg per hour, which, by the 13th of June had been increased to 9 mg per hour. On the 7th of June we did an epidural test block done at the Alaska Surgery Center Pain Unit with 2 cc of Fentanyl epidurally which provided her significant pain relief.

It was decided that an epidural catheter should be placed as soon as possible and constant infusion of epidural Morphine begun. On June 13th, Dr. Chandler placed a DuPen long term epidural catheter under sterile conditions with the patient awake using I.V. sedation and monitored anesthesia. The catheter was place at the L-2,3 level in a cephalad direction, the tip at about T-10. An initial bolus of 5 mg of Duramorph was given at the time of surgery. The patient then received 1 mg of Duramorph per hour for the ensuing 24 hours. She was admitted for 23 hour observation at the Alaska Surgery Center and determined to be in adequate pain control with no respiratory distress and was sent home. Her pain was controlled with epidural Duramorph for the remainder of her illness. All other narcotics were discontinued at the time her epidural catheter was inserted. On the 20th of June, a separate I.V. was inserted for the administration of Versed for anxiety. She tolerated this drug combination well and was able to converse, read and was lucid essentially through the remainder of her illness. The epidural Morphine was increased from the original 1 mg per hour to 20 mg per hour by the 5th of July when the patient died. Because of lack of peripheral veins, in the last week of her life a Groshong central venous catheter was placed to give the Versed.

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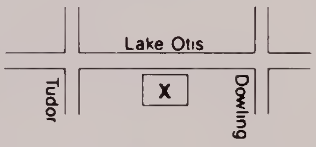

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BRIEF SUMMARY

Contraindications: Severe LV dysfunction (see *Warnings*), hypotension (systolic pressure < 90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no pacemaker is present), 2nd- or 3rd-degree AV block (if no pacemaker is present), atrial flutter/fibrillation with an accessory bypass tract (eg, WPW or LGL syndromes), hypersensitivity to verapamil.

Warnings: Verapamil should be avoided in patients with severe LV dysfunction (eg, ejection fraction < 30%) or moderate to severe symptoms of cardiac failure and in patients with any degree of ventricular dysfunction if they are receiving a beta-blocker. Control milder heart failure with optimum digitalization and/or diuretics before Calan SR is used. Verapamil may occasionally produce hypotension. Elevations of liver enzymes have been reported. Several cases have been demonstrated to be produced by verapamil. Periodic monitoring of liver function in patients on verapamil is prudent. Some patients with paroxysmal and/or chronic atrial flutter/fibrillation and an accessory AV pathway (eg, WPW or LGL syndromes) have developed an increased antegrade conduction across the accessory pathway bypassing the AV node, producing a very rapid ventricular response or ventricular fibrillation after receiving I.V. verapamil (or digitalis). Because of this risk, oral verapamil is contraindicated in such patients. AV block may occur (2nd- and 3rd-degree, 0.8%). Development of marked 1st-degree block or progression to 2nd- or 3rd-degree block requires reduction in dosage or, rarely, discontinuation and institution of appropriate therapy. Sinus bradycardia, 2nd-degree AV block, sinus arrest, pulmonary edema and/or severe hypotension were seen in some critically ill patients with hypertrophic cardiomyopathy who were treated with verapamil.

Precautions: Verapamil should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of overdosage. Verapamil may decrease neuromuscular transmission in patients with Duchenne's muscular dystrophy and may prolong recovery from the neuromuscular blocking agent vecuronium. It may be necessary to decrease verapamil dosage in patients with attenuated neuromuscular transmission. Combined therapy with beta-adrenergic blockers and verapamil may result in additive negative effects on heart rate, atrioventricular conduction and/or cardiac contractility; there have been reports of excessive bradycardia and AV block, including complete heart block. The risks of such combined therapy may outweigh the benefits. The combination should be used only with caution and close monitoring. Decreased metoprolol clearance may occur with combined use. Chronic verapamil treatment can increase serum digoxin levels by 50% to 75% during the first week of therapy, which can result in digitalis toxicity. In patients with hepatic cirrhosis, verapamil may reduce total body clearance and extrarenal clearance of digitoxin. The digoxin dose should be reduced when verapamil is given, and the patient carefully monitored. Verapamil will usually have an additive effect in patients receiving blood-pressure-lowering agents. Disopyramide should not be given within 48 hours before or 24 hours after verapamil administration. Concomitant use of flecainide and verapamil may have additive effects on myocardial contractility, AV conduction, and repolarization. Combined verapamil and quinidine therapy in patients with hypertrophic cardiomyopathy should be avoided, since significant hypotension may result. Concomitant use of lithium and verapamil may result in a lowering of serum lithium levels or increased sensitivity to lithium. Patients receiving both drugs must be monitored carefully. Verapamil may increase carbamazepine concentrations during combined use. Rifampin may reduce verapamil bioavailability. Phenobarbital may increase verapamil clearance. Verapamil may increase serum levels of cyclosporin. Concomitant use of inhalation anesthetics and calcium antagonists needs careful titration to avoid excessive cardiovascular depression. Verapamil may potentiate the activity of neuromuscular blocking agents (curare-like and depolarizing); dosage reduction may be required. Adequate animal carcinogenicity studies have not been performed. One study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. Pregnancy Category C. There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor, and delivery only if clearly needed. Verapamil is excreted in breast milk; therefore, nursing should be discontinued during verapamil use.

Adverse Reactions: Constipation (7.3%), dizziness (3.3%), nausea (2.7%), hypotension (2.5%), headache (2.2%), edema (1.9%), CHF, pulmonary edema (1.8%), fatigue (1.7%), dyspnea (1.4%), bradycardia: HR < 50/min (1.4%), AV block: total 1°,2°,3° (1.2%), 2° and 3° (0.8%), rash (1.2%), flushing (0.6%), elevated liver enzymes. The following reactions, reported in 1.0% or less of patients, occurred under conditions where a causal relationship is uncertain: angina pectoris, atrioventricular dissociation, chest pain, claudication, myocardial infarction, palpitations, purpura (vasculitis), syncope, diarrhea, dry mouth, gastrointestinal distress, gingival hyperplasia, ecchymosis or bruising, cerebrovascular accident, confusion, equilibrium disorders, insomnia, muscle cramps, paresthesia, psychotic symptoms, shakiness, somnolence, arthralgia and rash, exanthema, hair loss, hyperkeratosis, macules, sweating, urticaria, Stevens-Johnson syndrome, erythema multiforme, blurred vision, gynecomastia, increased urination, spotty menstruation, impotence.

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An Interview: Louis W. Sullivan, M.D.

In response to an invitation from Senator Ted Stevens, Dr. Louis W. Sullivan, Secretary of Health and Human Services visited Alaska during the week of August 11, 1990. Ray Schalow, ASMA Executive Director and Chairman of the Health Care Coalition (HCC) of Alaska, was asked by Senator Stevens to co-sponsor Dr. Sullivan's visit. During his visit, Dr. Sullivan agreed to an interview for *Alaska Medicine* as did Dr. Gail Wilensky, Administrator of HCFA. Dr. David E. Johnson of Ketchikan, prepared the interview questions for Dr. Sullivan and Dr. Wilensky. Dr. Wilensky's interview will appear in the December issue of *Alaska Medicine*.

Q: What is the administration's plan for the Indian Health Service in Alaska? What opportunities for cooperative efforts, shared facilities, etc., are there with the Indian Health Service, Department of Defense installations and the private sector?

A: We will continue to fulfill the federal responsibility to the Alaska Native population by providing it a health services program as an integral part of Indian Health Service (IHS) health programs.

We expect the IHS relationship with community hospitals to continue. The Alaska Native Health Clinic will maintain its location and relationship with the Fairbanks Community Hospital. IHS funded inpatient services are provided in Fairbanks Memorial Hospital by Tanana Chiefs Corporation/IHS physicians and by medical specialists from the private community in Fairbanks.

The Alaska Native Medical Center (ANMC) in Anchorage purchases many services from the private sector with its contract care funds. These include contracts for neonatal intensive care and burn unit care from Providence Hospital, and adult medivac services and overflow ICU care from Humana Hospital. These kinds of relationships will continue after the opening of the new ANMC.

The IHS and our uniformed service medical facilities have had long-standing cooperative relationships in Alaska. For example, the ANMC procures some of their nuclear medicine studies at Elmendorf Air Force Base Hospital and ANMC thoracic surgeons have assisted at the same hospital. the directors of these hospitals meet regularly. Bassett Army Hospital physicians have filled in at Barrow during times of critical physician shortages and the Coast Guard assists with emergency medical evacuations in Southern Alaska, among other examples of cooperative efforts.

Q: What policy initiatives does the Administration plan to encourage minority enrollment in medical and nursing schools?

A: In keeping with our commitment to increase access to health care services for minority populations, our FY 1991 budget request includes an additional $107 million for activities providing health care or training in health professions for minorities.

The $107 million includes $12 million for faculty and tuition assistance in minority health professions schools, $5 million for grants to communities and institutions for recruitment and educational support of health professions, and $55 million for the National Health Service Corps (HHSC) to recruit disadvantaged and minority health professions students to practice in underserved areas. The NHSC funds will support federal and state loan repayment agreements as well as scholarships.

Q: What does the Administration propose as appropriate federal action regarding student loans and grants for medical and nursing students where there are economic barriers to education?

A: As with all federally supported student loans and grants, priority for loans and grants for medical and nursing students is given to the economically disadvantaged. This has been the policy since such assistance was first initiated and it has been supported by administrations of both parties including the Bush Administration. I cited some specific initiatives for the benefit of economically disadvantaged minority students in the previous question.

Q: What can be done to address the shortage of and maldistribution of physicians, particularly primary care physicians?

A: National Health Service Corps scholarships are awarded to medical students interested in primary care. On completion of medical training, scholarship recipients are required to serve in health manpower shortage areas. Our Health Resources Services Administration (HRSA) also supports the training of primary care physicians by awarding funds for the training of physicians in family medicine, general internal medicine, and general pediatrics.

In addition, HRSA seeks medical school faculty members for special training programs which enable them to serve as role models for entering students in the hope that careers in family practice will be encouraged. Medical school programs that motivate students to consider the underserved areas of medicine are also funded by HRSA.

Q: What can be done to address the shortage of practicing patient care nurses in hospitals, particularly rural and inner city hospitals?

A: Health Resources Service Administration (HRSA) - financed studies reveal that nurses want more autonomy, increased respect, stable shifts, and more time for hands-on primary care. Strong nursing leadership has been shown to be a motivating force in encouraging nurses to remain in the field. HRSA's Division of Nursing is funding innovative practice models, including two at the University of Arizona and the University of Rochester. HRSA also funds programs aimed at recruitment and retention of rural health nurses.

Q: Is there any appropriate role for the Federal Government in addressing the liability problems confronting our profession in particular and society in general?

A: An August 1987 report issued by a special HHS medical liability task force recommended the enactment of strong tort reform measures as a solution to the liability crisis. The report recommended a federal leadership role in the area that has been traditionally the responsibility of the states.

Now, as the Federal Government is increasingly called upon to remedy health care cost and access problems, the Bush Administration and the Congress are examining ways to address problems caused by poor outcomes in health care which often result in claims for medical malpractice.

Many solutions to the complex liability problem are being considered. Among these are various incentives to states that limit awards for pain and suffering, limit statutes of limitations, and create arbitration panels to hear disputes and award damages. Other ideas include ways of making the current system for resolving malpractice claims more efficient, such as the AMA's model legislation for state reform of handling medical malpractice cases. Some states are experimenting with a no-fault liability system.

Of course, the surest way to reduce malpractice claims is to reduce poor outcomes. Within HHS, there are various initiatives addressing health care quality assurance and risk management. The newly created Agency for Health Care Policy and Research's Medical Treatment Effectiveness Program has been developed to enhance our understanding of the effects of variations in health practices on patient outcomes.

Q: We have been involved in some beginning exchanges with physicians in the Magadan Region in Far-Eastern Russia. Are there federal resources that could be brought to this exchange?

A: I am pleased to see the broadening of contacts between health professionals and citizens of the United States and the Soviet Union. It offers further evidence of the new openness or "glasnost" in the Soviet Union. Federal funding, through HHS, of health-related exchanges is limited generally to collaborative research of direct relevance and interest to such agencies as the National Institutes of Health, Centers for Disease Control, and the Indian Health Service. Research proposals would need to comply with the normal application and competitive review procedures established by these agencies. I would encourage the private sector to assist in these exchanges.

Q: Tobacco remains the most common substance of abuse in our entire population, from teenagers through retirees. What further steps can we take to discourage folks from beginning to smoke?

A: Approximately 90 percent of smokers start smoking before the age of 20, making young people by far the largest group of new smokers. HHS has taken several actions in the effort to curb tobacco use among young people. In May 1990, we released a "Model Sale of Tobacco Products to Minors Control Act: and sent a copy to each state governor in an attempt to encourage them to strengthen tobacco access laws where they exist and to adopt such legislation where there is none.

A report prepared by the HHS Inspector General, "Youth Access to Cigarettes," found that despite legislation in 44 states to control minors' access to tobacco, some 3 million young people under the age of 18 consume 947 million packs of cigarettes each year. In 1989, only 32 vendor violations for cigarettes sold to minors were reported by the states.

Further HHS efforts to discourage smoking include conducting national public information and education programs, targeted both to adults and to young people. Special emphasis is placed on teenagers, pregnant women, and smokers who are seeking help in quitting. Campaign messages are delivered to the public via television, radio, and other media and are developed in cooperation with other government agencies, voluntary organizations, and professional groups.

Q: The Administration has opposed federal funding for both abortions and birth control. American society has one of the highest rates of teenage pregnancy among the developed countries, particularly among some minority groups. What is the Administration proposal to address the problem of pregnancy in adolescents?

A: Our Administration has not opposed funding for family planning. Overall federal expenditures for family planning have more than doubled over the past ten years. The major programs are the Medicaid family planning program (90 percent federal financial contribution) and the Title X family program which serves an estimated 4 million people. Family planning support is provided also by the Maternal and Child Health Block Grant program and by the Social Services Block Grant program.

Our Department is working to address the teen pregnancy problem not only through our family planning and maternal and child health programs, but through other innovative approaches that attempt to get at the root of the problem. The Adolescent Family Life program, for example, is funding demonstration projects which provide prevention services designed to address the underlying problems that lead adolescents into early sexual activity and out-of-wedlock pregnancy, as well as care services for teens who are pregnant. This program has a strong commitment to strengthening families and promotes adoption as a positive option for unmarried pregnant teens.

Q: AIDS can be addressed effectively only by prevention. Prevention is possible only with responsible individual behavior. Research seems to show that IV drug abusers and sexually active adolescents have not changed their behavior substantially. What should the Federal Government do to encourage responsible individual behavior regarding AIDS?

A: The Federal Government can best encourage behavior that reduces the risk of HIV infection through a vigorous, credible, and effective education campaign. With the 1991 budget request for AIDS, the Public Health Service will have spent about $1.5 billion in total on AIDS prevention education activities. While early efforts focused on large metropolitan areas, the Centers for Disease Control now provides financial and technical assistance in support of comprehensive prevention programs to all 50 states.

Evidence is emerging which suggests that AIDS prevention education programs are having a positive impact in changing behaviors that place people at risk of HIV infection. Nationally, for example, syphilis rates for all white males declined 68 percent between 1982 and 1989, reflecting safer sexual behavior among this large population.

Similarly, federal education efforts in the war on drugs have contributed to a downward trend in overall drug use in America. Overall drug use declined by some 37 percent between 1985 and 1988, according to the National Institute on Drug Abuse.

Hopefully, some readers of Alaska Medicine will recall that when I addressed the Alaska Drug Summit in August, I stressed the need for developing a stronger sense of individual responsibility -- a culture of character -- as essential to our prevention efforts. In the final analysis, it will be greater acceptance of individual responsibility which will end the sort of behavior that places people at risk of HIV infection.

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AIR FORCE

History of Medicine in Alaska

M. Walter Johnson, M.D.

Walter Johnson was not born in Alaska but by the age of twenty had become so immersed in it that it seems that he should have been.

His grandparents were homesteaders in Nebraska. His father was a farmer born in a sod house. His mother came from a background of ministers and physicians.

Walter attended a one-room schoolhouse with one teacher and twenty pupils. In the winter, they huddled around a coal stove. Classes went in turn to the front for instruction. By the 6th grade, he had mastered the curriculum. Before going to high school he took time out to work on the farm.

Since the depression and the drought made farming difficult, the family moved to Lincoln. Walter graduated from high school at the age of nineteen, winning a Regent's Scholarship to the University of Nebraska. An advantage of living in Lincoln was the library which provided unlimited reading material and intellectual stimulation.

Walter became a pen pal with a youth from Alaska who was taking high school correspondence courses from Nebraska. He acquired one of the first multiple speed bicycles from England. In 1941, Walter rode his bike to the west coast except for a couple of hundred miles in a truck. He slept on the ground and estimates that it cost him 35 cents a day to travel.

From Seattle, he went by Alaska Steamship to Juneau where he met his pen pal and his family - the Bolyans. They took him to the west coast of Chichagof Island where they operated a gold mine. He worked in a nearby gold mine for the summer. In the fall he made his way back to Seattle by catching rides on fishing boats. He spent a year at the University of Nebraska.

The next summer he returned to Southeast Alaska where, after procuring his seaman's papers, he served as cook on the fishing boat of Andy Gunderson. Since this fisherman was campaigning for the territorial senate, they visited all the villages in the area. Gunderson was elected.

Walter then took the Alaska Steamship to Valdez where he found a ride to Fairbanks. He enrolled in the University of Alaska becoming one of the hundred and fifteen in the student body.

During WWII the military programs for premed students were not offered in Alaska, so he went into basic training at nearby Ladd Field. After boot camp he was assigned by request to the hospital laboratory at Ladd. Here he was to spend the war on an overseas assignment which was a move of only four miles.

While in the service, Walter met Bill English who was born in Wiseman and who had spent his early childhood there, but had attended schools in California. His mother was an Eskimo. Bill introduced Walter to Wiseman which he began to visit whenever possible.

The war had ended gold mining in the area but since it was hoped that mining might resume, Bill and Walter bought the Wiseman trading post from Bill's father. In any event, they hated to see the trading post close. Walter took time off from the university to spend a winter in Wiseman, working for the road commission. In Wiseman each summer for seven years, he had ample opportunity to hike and explore the Brooks Range. In preparing a lecture on Wiseman recently, he recalled the names of all 42 people living in the 15000 square mile area in 1946.

After assisting one of Alaska's better known guides on a bear hunt, he took the Alaska guide examination. In 1948, he was issued a guiding license and the same year he received a B.S. degree in Anthropology and Biology. He was named the outstanding senior student.

He was in a dilemma as to whether to pursue a career in medicine or in the outdoors. So he took time out to consider, hunting and working on the airfield at Wiseman. He decided that there were no jobs for biologists in the Territory and having thus chosen medicine, he went Outside to take medical school entrance examinations and was accepted at the University of Nebraska.

During the first two summers in medical school he returned to Wiseman. It was a long trip: by train to Seattle, by DC-3 to Fairbanks, and by small airplane to Wiseman. During the third summer he worked at the University Hospital during a polio outbreak.

After graduation from medical school he spent 2 1/2 months assisting two physicians with busy practices in a small town in northern Nebraska. They alternated taking vacations and Walter learned a great deal especially from one of them with unusual insight into dealing with people. Then he spent September and October camping, hiking and hunting in the mountains on the way to Spokane, Washington where he interned at Sacred Heart Hospital. On his weekly day off, he assisted an obstetrician in his office, earning his first money as a doctor.

Next he accepted a position under civil service on the staff of the newly opened Bethel Hospital along with Dr. Harriet Jackson who had arrived some months earlier.

Tuberculosis was cresting on the Kuskokwim but because of Isoniazide the outlook was improving. Walter vividly recalls a ten year-old boy moribund with Tuberculosis meningitis in extreme opisthotonus, a condition usually seen only in physical diagnosis text books. Isoniazide was injected, and dramatically, the patient responded and was running around the ward in a few weeks.

The hospital census generally was in the forties but once it reached sixty-five. Outpatients seemed numberless since the hospital also served many villages. There was constant struggle with the radio because of poor reception, and untrained personnel. Often these were school teachers who had received only a few hours training before going into a village.

Anesthesia was administered by a dentist, who was a borderline cardiac patient.

On New Year's Eve, 1955, during an emergency C-section, a steam pipe inside a wall burst blasting steam through the sheet rock and into the operating room. The engineer cut off the steam but fortunately, not the lights.

Spirits at the hospital were uplifted by visits by specialists from Anchorage: Drs. John Tower, pediatrics; William Mills, orthopedics; and Joseph Shelton, EENT. These consultants worked long hours holding clinics. Dr. Philip Moore toured western Alaska seeking patients with complications of bone Tuberculosis for treatment in Sitka. Dr. Earl Albrecht, Territorial Commissioner of Health, had recruited Dr. Moore who pioneered the use of streptomycin for Tuberculosis patients undergoing bone surgery. Dr. Philip Moore died on June 17, 1990 at the age of 86 in Sequim, Washington.

Dr. Johnson made many field trips to the lower Yukon which had not been visited by a physician since the 1940s when the Territorial boat "Yukon Health" made trips up the river. There were many undiagnosed and unattended problems: Pott's Disease, dislocated hips, visual defects and endless dental caries. Walter liked to spend about a week in a village and end with a community dance. He found it so easy to interact with the Native people that twenty to thirty years later there is still mutual recognition.

In the spring of 1956, Walter Johnson decided that itinerant visits by physicians were no solution to the problem of inadequate health care. In a suggestion for formal training for health aides, he wrote in a letter to the Director of the Alaska Native Service in Juneau: "It is not a question of whether the villager shall be treated by completely qualified medical personnel or persons with less than full qualifications, but a question of whether they shall be treated by persons with limited qualifications or go untreated altogether."

While at Bethel, Walter was commissioned in the U.S. Public Health Service after which he was sent to the PHS Hospital in Chicago. He served as a resident in internal medicine at the prestigious USPHS Hospital in Boston where he remained for three years. Founded as the Boston Marine Hospital in 1798 by President John Adams to serve the merchant marine, it has operated continuously since then.

He served for two years at the PHS Hospital in Seattle and one year in Memphis. Between these assignments he was with the Peace Corps, headquartered in Washington, D.C., conducting a medical survey in Ethiopia and Somali.

After returning to Alaska in 1963 he served as Chief of Medicine at the Alaska Native Medical Center and later was Medical Director, followed by six years in the Community Health Aide training program as Director and Instructor.

In 1967 he served temporarily as a tuberculosis consultant for the Peace Corps in Istanbul and intermittently until 1976, as consultant and co-director of the Tunisia Tuberculosis Project under the federal Food for Peace program.

His experience in Tunisia proved the efficacy of the concept of training local health aides to improve life in the villages. Grant money was used to buy German microscopes and Tunisian village youths were trained to examine sputum to identify tuberculosis. This was tremendously efficient and the incidence of tuberculosis declined.

After retirement from the Public Health Service, he continued working at the University of Alaska with the Community Health Aides Program which began in Alaska out of local need and on a voluntary basis. As early as 1964 three public health physicians gave rudimentary training to aides at Bethel, Mt. Edgecombe and Kotzebue. In 1968 the Alaska Community Health Aide Program was established by Congress. Through this act federal funds were provided for the training and payment of health aides. The legislation designated one aide per isolated village and one for each three hundred persons in larger communities.

The Community Health Aide concept stressed permanence of residence as fundamental, and the desirability of fluency in the local dialect. Training programs evolved in Anchorage, Bethel and Nome.

In 1973 it was decided to bring the three programs together to coordinate and standardize training. An important accomplishment was the writing of a health aide manual, "Guidelines to Primary Health Care in Rural Alaska, 1976" by Joseph Whittaker. A statewide uniform health aide curriculum was developed jointly by the three training program staff members.

Training periods away from the villages are kept short - three sessions of four, three, and three weeks. Between sessions, the CHA practices in her village under telephone or radio supervision by a physician. On-site training is given by visiting professionals: the physician making village visits, a mid-level practitioner, and the public health nurse.

The Community Health Aides have met a need admirably. Walter cites the example of an aide identifying Addison's Disease and making the appropriate referral. The program has had a high degree of acceptance by physicians and is held in great esteem by professionals concerned with arctic health. At the May 1990 Symposium on Circumpolar Health held in Whitehorse, Yukon Territory, it was praised as a sterling example of the possible.

Walter Johnson has been married twice. He has three sons, one daughter, and a granddaughter. His most rewarding relationship, however, is with Anne Wieland, an Anchorage science teacher of note who is involved with the Trailside Discovery Camp which provides outdoor education.

Walter knew that it was difficult to keep up professionally in Alaska without a medical library. When he became involved in getting a library at the Alaska Native Medical Health Center, Ursula Strash was prevailed upon to become librarian before many materials were obtained essentially becoming a librarian sans a library. Walter got the Regional Medical Program to fund what would become the Alaska Health Sciences Library.

From the beginning the library had the commitment to serve all health professionals around the state. It was linked to the Regional Medical Library at Seattle and the National Library of Medicine in Maryland and later to MEDLINE. By this time nearly eighty percent of the physicians in the state were making use of the library. A unique feature was the "table of contents" service whereby health workers could subscribe to a periodic mail-out of the table of contents of selected journals.

In 1977 the Alaska Health Sciences Library moved to the University of Alaska, Anchorage Consortium Library and is now funded principally by the State of Alaska.

Walter Johnson retired in 1987. In 1989 the University of Alaska conferred upon him an Honorary Degree of Doctor of Public Service citing him for being the first University of Alaska graduate to receive a medical degree and return to the Territory; for his work in tuberculosis; for his effort in founding the Alaska Health Science Library, and for creating the Alaska Community Health Aide Program.

Walter had his first garden at age eight. As a youth he went to sea. Now sixty years and a medical career later, life for him has come full circle. He summers in Homer where he gardens and plants trees on his homestead and offers a charter boat service on Kachemak Bay.

Gwynneth Gminder Wilson
Alaska State Medical Association Auxiliary

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American Society for Circumpolar Health

The American Society for Circumpolar Health sponsored an "International Workshop on Alveolar Hydatid Disease" (Echinococcus multilocularis) in collaboration with: the Centers for Disease Control, Indian Health Service, World Health Organization, and International Union for Circumpolar Health. The meeting was held in Anchorage, June 7-8, 1990 and was attended by over 60 invited participants with experience and expertise from areas endemic for E. multilocularis infection. These areas include: Europe, USSR, China, Japan, Canada and the U.S. A broad range of topics: epidemiology, immunology, molecular biology, diagnosis, treatment, prevention and control were addressed. Recent advances in research were presented and areas requiring research in the future were identified. Copies of the program and abstracts are available on request from conference organizers: Anne Lanier, M.D., M.P.H., Arctic Investigations Program, CDC, Anchorage, Alaska (271-4011) or Peter Schantz, V.M.D., Ph.D., Division of Parasitic Diseases, CDC, Atlanta, Georgia, 30333.

A group sponsored by the Alaska-Siberia Medical Research Program led by Dr. Ted Mala of the University of Alaska - Anchorage traveled to Magadan, USSR during July 21 through August 4, 1990. The groups consisted of 32 participants currently working in the areas of medicine, nursing, psychology, physiology, injury and other health-related fields. Participants were spread out among different communities. A condition of travel was that all participants file reports on their findings and impressions. A composite of these reports will be published in a forthcoming issue of Alaska Medicine. The Alaska-Siberia Medical Research Program is already receiving applications for next year's travel. For more information contact Dr. Ted Mala, 786-4746.

Inuit Studies Conference

The Seventh Inuit Studies Conference, the first one to be held in the USA, convened at the University of Alaska Fairbanks on August 19 through 24, 1990. The conference theme of "Looking to the Future" allowed for examination of issues from historical, contemporary, and future perspectives for their impact on the lives of the Inuit.

The Inuit Studies Conferences, held biannually since 1978, are the forums of the Association of Inutisiutiit Katimajiit, Inc., a non-profit organization devoted to the study and promotion of Inuit (Eskimo, including Aleut) culture, language, and society. The conferences deal with fundamental and applied issues pertaining to the Inuit in the realms of anthropology, sociology, linguistics, history, health, education, politics, economics, and related social and cultural topics. The first five convenings of the Inuit Studies Conference took place in Canada, and the sixth in 1988, at the Institute of Eskimology in Copenhagen, Denmark.

There were over 300 participants including 55 members who traveled from the Soviet Union to attend. The conference is endorsed by the International Union for Circumpolar Health. Dr. J.P. Hart Hansen, President of the IUCH, chaired the health section entitled, "Health, Healing and Medical Issues."

Northern Regions Conference

The Third Northern Regions Conference is an opportunity to build upon a relationship of cooperation and understanding between Arctic and North Pacific nations. The conference will be held this September 16th through 20th in Anchorage, Alaska.

Leaders and residents of the Arctic and North Pacific will have an opportunity to meet and explore issues of regional development and cooperations. The conference is organized into three parts; beginning with a Governor's Summit on the Environment, followed by a Business Forum on industrial and resource development and Workshops on political and social issues of northern living.

This conference builds upon the efforts of the two previous Northern Regional Conferences which were held in Japan (1971) and Canada (1979). The First Northern Regions Conference was organized by Naohiro Dogakinai, Governor of Hokkaido, Japan.

It focused on the common economic and cultural experiences of northern countries. This first conference was primarily attended by government officials from Japan, Canada, Finland, Norway, Sweden and the United States.

The Second Northern Regions Conference was hosted by Alberta, Canada. It focused on ways to improve the environment, expand resource development, and facilitate urbanization in northern regions. In addition to the six original participants, South Korea and the People's Republic of China attended the conference.

"Cooperation in a Changing World" is the theme of the Third Northern Regions Conference, sponsored by Steve Cowper, Governor of Alaska. The scope of the third conference has expanded to include participants from eleven nations and meet the demands of world changes. For the first time there will be representatives attending from Denmark/Greenland, Iceland and the Soviet Union.

The United States Ambassador to Denmark, Keith Brown will give a luncheon keynote address on Monday, September 17. Ambassador Brown is the head of the United States mission to Denmark and the home rule governments of Greenland and the Farol Islands. At the conference, Ambassador Brown will present the official U.S. Arctic Policy in areas such as economics, environment, science, security, and culture. Ambassador Brown will also comment on trends and developments in the Arctic, particularly as they relate to Alaska and Greenland.

The conference is co-sponsored by the International Union for Circumpolar Health and there will be a workshop on Circumpolar Health. The President of the IUCH, Dr. J.P. Hart Hansen will be participating along with members from the American Society for Circumpolar Health.

Arctic Science Conference

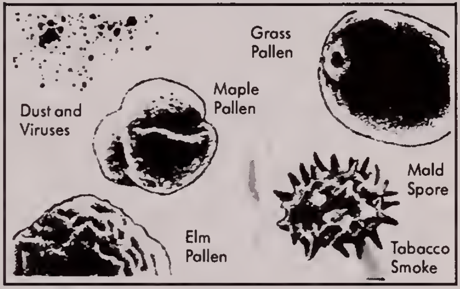
The 41st Arctic Science Conference will be held during October 8 through 10, 1990 in Anchorage, Alaska. The theme for the conference is "Circumpolar Perspective" and will serve to illustrate both the international scope of Arctic research and circumpolar interests of arctic scientists. This year's conference, like those in the past, is organized by the Arctic Division of the American Association for the Advancement of Science (AAAS). Traditionally, a primary purpose of the conferences has been to provide an annual forum for multidisciplinary discussions of arctic science and of its social and policy implications. The conference this year is organized so that there will be discussions both in sessions with special foci and in general sessions for contributed papers in any field.

A second, important purpose of the conference is to help with the education of future arctic scientists by serving as a resource for science teachers. The conference is scheduled so that the first day coincides with an In-Service day for the science teachers of the Anchorage School District. A third purpose is to provide facilities for collateral meetings of affiliated societies.

This year's conference will hold a special session on medical sciences. Dr. John Middaugh will chair the session on "Circumpolar Health Issues."

The American Society for Circumpolar Health is scheduled to hold their Annual Meeting during the conference. This will be a luncheon meeting on October 9th at the Sheraton Hotel (host facility). Many members are expected to attend.

VISITORS!



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ALASKA
PUBLIC HEALTH ASSOCIATION

President's Report
August, 1990

by Brian Saylor, Ph.D., M.P.H.

Health and Human Services Secretary Louis Sullivan urged Alaskans to meet the challenges of preventable diseases related to injuries, smoking, inadequate nutrition and exercise, and drug and alcohol abuse during the annual health care "summit" on August 14 at the Anchorage Hilton. The Alaska Public Health Association, as part of the Health Care Coalition, helped organize and sponsor this important event which also included Senator Ted Stevens as a key participant.

The Secretary focused his remarks on many of the public health issues that have been of great concern to our Association. The problems resulting from these and other conditions common in our region, compounded by vast distances and geographic barriers, require a strong "partnership among federal, state and local governments and Native health corporations." The Secretary was impressed by the strong working relationships among these groups in Alaska.

Sullivan also sees health care costs as a major challenge to his administration. He said that the country can no longer tolerate the high cost of medical care. "The time for answers is surely at hand", he said. He has committed his staff to prepare a comprehensive review of federal health care policies for President Bush by early next year.

The Secretary's comments were echoed by Dr. Gail Wilensky, the new head of the Health Care Financing Administration. She said that while the country was getting closer to consensus on the nature of the problem of rising health care costs, there is little consensus on solutions. According to Dr. Wilensky, "a single unified insurance plan is not really in our future." She prefers to continue to rely on the existing synthesis of public and private sector service provision and financing. She stated that this would retain the very best in American medicine.

Secretary Sullivan said the medically indigent are of particular concern. But so far, the Bush administration has "resisted pressure to prime the federal pump" through using new federal funds to pay for the medical costs of the uninsured. Medicare and Medicaid are already in place and can be used as the vehicles for addressing the problem, according to the Secretary.

Alaskan health care providers also met with Senator Stevens and his staff to discuss priority Alaskan health care issues. A common theme was the value of prevention. The success of the Hepatitis B vaccination program and the AIDS prevention program were examples of the returns to the people of Alaska from investments in prevention. Senator Stevens mentioned his efforts to establish and fund a new Center for Injury Control with CDC. This effort is especially important to Alaskans in view of our high injury morbidity and mortality. Important work deserving of federal attention include the development of water and waste water systems which can be effectively maintained, and encouraging programs which empower communities to assume more control over their own health and welfare. These meetings provided Alaskan public health professionals a unique opportunity to discuss critical health issues with these key policymakers.

Another area of interest for health professionals is the recent activity of the Alaska Health Care Coalition (HCC). The HCC developed a summary of important health issues which is outlined below.

Health Issues for Gubernatorial Candidates

The following summary of important health issues was developed by the Health Care Coalition. The Coalition (HCC) represents the professional interests of over 15,000 Alaskan health care providers. The Alaska Public Health Association was one of the original members of the coalition.

The HCC believes that it has a responsibility to educate Alaskan leaders about pressing health care issues, and to be available as a continuing resource as those issues are discussed and resolved. A complete discussion of these issues was circulated to each candidate for Governor of Alaska, and has quickly become the principle reference document on health care issues for aspiring candidates.

Prevention Issues

- Maintain aggressive immunization programs.
- Expand injury prevention and control efforts.
- Expand education in dental hygiene.
- Enhance health education activities to include chronic and behavior-related conditions such as alcohol abuse, child abuse and AIDS.
- Encourage demonstration programs to more effectively address the problems of the medically indigent.
- Support activities which protect our environment.
- Design rural water and sanitation systems to ensure proper maintenance.
- Remain vigilant about the transportation of hazardous wastes and environmental integrity.

Access Issues

- Encourage demonstration programs to more effectively address the problems of the medically indigent.
- Facilitate the orderly transfer of health care delivery responsibilities to Native Regional Health Corporations.
- Renovate or replace health facilities that fail to meet State standards for health and safety.

Health Care Delivery Systems Issues

- Determine the State's health requirements.
- Support the Alaska Public Health Policy Task Force.
- Develop educational programs to train needed professionals and create incentives to recruit and retain manpower in underserved rural areas.
- Quantify shortages in certain health provider professions.
- Strengthen the occupational health and safety system.

Cost Issues

- Expand the mission of the Universal Health Care Task Force to address innovative methods of reducing the cost of care and the number of uninsured.

Leadership Issues

- Strengthen the health policy leadership by requiring stringent qualifications for the Commissioner of Health and Social Services and for the Director of the Division of Public Health.

- Develop plans to define the State's responsibility for the provision of direct services.

ALPHA BROWN BAG LUNCHES

Brown bag lunches are held during the months of September through May in the communities of Anchorage, Bethel, Fairbanks, Juneau, and Ketchikan. The program provides an opportunity for individuals who are interested in public health issues to gather for discussion and continuing education.

Anchorage
Coordinator Bert Hall 343-6718
3rd Thursday of the month at the Municipality's Health Department
September 20 - 1990 Medical Expedition to Magadan/Chukotka

Bethel
Coordinator Corlis Taylor 451-2331
2nd Friday of the month - location and topic to be announced.

Fairbanks
Coordinator Cheryl Kilgore 451-2940
2nd Tuesday of the month, Medical Dental Arts Building Conference Room
September 11 - A Report on the 8th International Congress on Circumpolar Health

Juneau
Coordinator Position is vacant - any volunteers?

Ketchikan
Coordinator Jeanne Ponti 225-9431
Date, location and topic to be announced

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Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

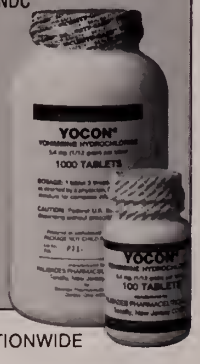
Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:
1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

Rev. 1/85



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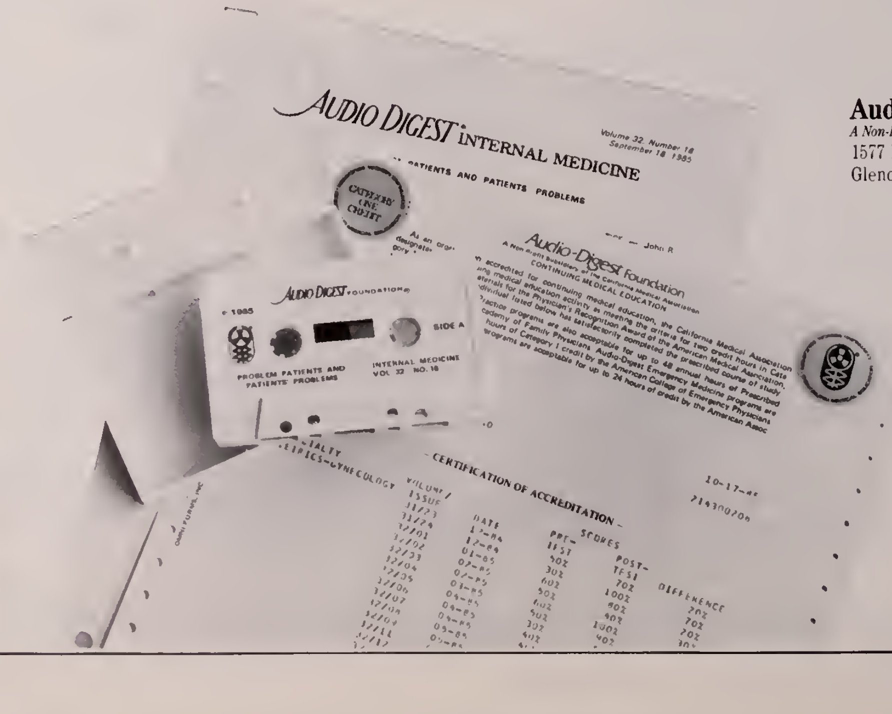

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Volume 32, Number 18
PATIENTS AND PATIENTS PROBLEMS
Audio-Digest Foundation
CONTINUING MEDICAL EDUCATION
PROBLEM PATIENTS AND PATIENTS' PROBLEMS
INTERNAL MEDICINE VOL 32 NO. 18
SIDE A
CERTIFICATION OF ACCREDITATION

GLIMPSES OF ALASKAN MEDICAL HISTORY

Edited by Robert Fortuine, M.D.

THE NOTEBOOK OF A SHIP'S DOCTOR (1816-17)

In 1815 Lt. Otto von Kotzebue set out from the Russian naval base at Kronshtadt for a round-the-world voyage. His principal task was to search out a navigable passage from west to east somewhere north of the Bering Strait, an area inadequately explored by Cook nearly four decades before. By the time the men of the brig *Riurik* returned home three years later, they had discovered and explored Kotzebue Sound--which of course did not provide the hoped-for route--and had also added to the map numerous islands in the central Pacific.

The ship's surgeon on the voyage was Dr. Friedrich Eschscholtz, an ethnic German like the captain. In contrast to many naval surgeons of the time, however, he was university trained and possessed a wide knowledge not only of medicine, but also of botany, zoology, geology, and other natural and biological sciences. He was apparently highly regarded by all who knew him and was an especial favorite of the young Kotzebue, who named a large bay in southeastern Kotzebue Sound in his honor. There the doctor had discovered what he called an "ice mountain," an impressive glacial remnant containing numerous fossil bones.

Eschscholtz wrote several sections of Kotzebue's official account of the voyage, including papers on rocks, butterflies, marine invertebrates, and a new species of monkey. But his principal contribution, excerpted below, was an essay on the health of the crew during their three-year voyage in tropical as well as arctic waters. Although his reflections on overindulgence in salmon might hold lessons for us today, of perhaps greater interest is his mention of worms in the flesh of the cod--quite possibly anasakids--and the care which the Aleuts showed in avoiding them. Syphilis had taken a terrible toll among the Aleuts at Unalaska around the time of Cook, but 40 years later it was, for unclear reasons, largely under control in this population.

Dr. Eschscholtz also accompanied Kotzebue on his second round-the-world voyage (1823-26), which briefly called at Sitka. He later became a professor at Dorpat, in present-day Estonia, and seems to have died in 1829.

"During our stay at Oonalashka, at so early a season of the year as April, when we had a constant succession of rain, snow, and storms, and it was necessary to work and to live in the open part of the ship, it was no wonder that catarrhal fevers, colds, coughs, and hoarseness should become almost general among the crew. Under such circumstances hoarseness is a complaint both difficult to cure and dangerous, because quinsey is much to be feared. A sailor who worked at the forge suffered complete hoarseness for four weeks, and even after he was cured, his voice was harsh for a whole year.

"When some weeks after our arrival salmon was caught in abundance, and the crew had it every day for dinner, frequent diarrhoeas ensued, either because the cook did not always boil the fish enough, or because they ate too much of it, after having been so long deprived of fresh provisions. At first I was for treating these, like usual cases of diarrhoea, arising from catching cold, not supposing the fish to be the cause, and administered slight stimulants and stomachics, upon which, however, head-aches and sickness at the stomach followed. Upon this I gave an emetic, on which the patients were immediately better, and some stomachic medicine put an end to the whole illness in a few days. . . .

"Till the salmon came in, the crew received daily a sufficient quantity of fresh cod; but nobody complained of intestinal worms, though the cod here abound in them, for which reason the Aleutians, who eat these fish raw, cut them into very thin slices to avoid these animals. . . .

"The frequent rheumatism in the joints, and particularly in the reins, and the attacks of gout, which many of the Russian fox-hunters, in the service of the Russian American Company were affected with, were generally considered as syphilitic symptoms. Among the many patients that I attended here, I could not, at this time, find any thing of a syphilitic nature. But I do not mean to say that syphilis has not yet reached this country; for how could this be possible, considering the numerous ships of various nations that have resorted hither since 1742? Nor do I know to what disorder I should ascribe a sunken nose. The patient, as he told me, had cured himself of syphilis, with fumigations of cinnabar: at present, being too much addicted to drinking rum, he was subject to gouty swellings in the arms in winter, and in the legs in summer.

"Nor was any of the sailors infected with this disease: it happened only that a small blister appeared, which soon dried off on the use of *Hydrargiri praecipitati rubri.*"

REFERENCE

Eschscholtz, Frederick. On the diseases of the crew during the three years of the voyage. In: *A voyage of discovery. . .*, by Otto von Kotzebue. 3 vol. [1821]. Reprint. New York: Da Capo Press, 1967. Vol. 2, pp. 333-336. .

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RESOLUTIONS

ADOPTED AT THE
ALASKA STATE MEDICAL ASSOCIATION ANNUAL MEETING
Fairbanks, Alaska

June 9, 1990

RESOLUTION 90-01
SUBJECT: Urging PROW/A to Publish Explicit Review Criteria

BE IT RESOLVED, that the Alaska State Medical Association strongly urges the Professional Review Organization of Washington and Alaska to explicitly state all outpatient and inpatient review criteria, in order that physicians be encouraged to practice high quality medicine and in order that both patients and physicians be spared needless paperwork and harassment in the pursuit of high quality medical care.

RESOLUTION 90-02
SUBJECT: Mandatory Seat Belt Law

WHEREAS, the use of seat belts has been shown to significantly decrease morbidity and mortality; therefore

BE IT RESOLVED, that the Alaska State Medical Association respectfully requests the Alaska State Legislature enact mandatory seat belt legislation.

RESOLUTION 90-04
SUBJECT: Mercury-Amalgam Fillings

WHEREAS, the possibility of mercury poisoning from amalgam fillings has been exaggerated and misrepresented, and

WHEREAS, the actual risk is infinitesimally small, and

WHEREAS, the supposed mechanism of absorption of mercury vapor from fillings is based on irrational premises, and

WHEREAS, many patients are wasting thousands of dollars on replacement of amalgam fillings by less durable ones, and

WHEREAS, no acceptable epidemiologic evidence supports the contention that amalgam fillings are harmful to patients, therefore

BE IT RESOLVED, that the Alaska State Medical Association recommends that patients not seek replacement of mercury amalgam fillings for the purpose of reducing body mercury load or mercury toxicity, since the actual risk of mercury poisoning is infinitesimally small.

RESOLUTION 90-05
SUBJECT: Diagnosis and Therapy of Putative Immune Deficiency

WHEREAS, the effects, if any, of environmental substances except infectious agents themselves upon immunity remain hypothetical, anecdotal, and unsupported by authoritative, corroborated documentation, and

WHEREAS, the role of irradiation, chemical exposures and other diseases in causing immune deficiency has been established through scientific studies and observations, therefore

BE IT RESOLVED, that the diagnosis of immune system deficiency (other than as described by the American Academy of Allergy and Clinical Immunology) in the absence of specific evidence of the deficiency, and the provision of therapy not shown to exceed placebo in efficacy, has no scientific basis and is unnecessary.

RESOLUTION 90-06
SUBJECT: Use of EDTA in treatment of atherosclerosis

WHEREAS, disodium ethylenediamine tetraacetic acid (EDTA) given intravenously binds to polyvalent metals, such as calcium, iron, copper and lead, forming water-soluble complexes which are excreted in the urine, and

WHEREAS, there is no convincing evidence that EDTA works in any other way, such as enzymatically or by reducing production of free superoxide or hydroxyl radicals, and

WHEREAS, EDTA is promoted for treating atherosclerosis by removing calcium from atherosclerotic plaques, despite the fact that calcium is only a minor component of plaques, and

WHEREAS, there are no controlled or prospective studies which show that atherosclerosis or its manifestations are ameliorated by use of EDTA, and

WHEREAS, intravenous EDTA can cause pyridoxine and zinc deficiencies, loss of digitalis effect, cardiac dysrhythmia, renal tubular necrosis and death, therefore

BE IT RESOLVED, that the use of EDTA for treatment of atherosclerosis has no scientific basis, and is medically worthless.

RESOLUTION 90-07

SUBJECT: Commitment to Modern Scientific Medicine

WHEREAS, the practice of medicine has changed over the centuries, from its earliest beginnings as shamanism, forklore and barber surgery; and

WHEREAS, the theoretical foundations upon which modern medicine is based were immeasurably strengthened by the development of the modern scientific method, and

WHEREAS, knowledge and intellectual discipline produced by the use of the scientific method can now be said to undergird the modern practice of medicine, and

WHEREAS, the ethical practice of modern medicine requires dealing honestly with patients and colleagues and advancing scientific knowledge, therefore

BE IT RESOLVED that the basis of modern scientific medical practice is encouraging every patient and family to follow a healthful lifestyle, with appropriate nutrition, exercise, sleep, behaviors, and emotional and spiritual renewal, and

BE IT FURTHER RESOLVED that the Alaska State Medical Association declares itself an organization committed to compassionate, competent and modern scientific medicine, and affirms its reliance on the modern scientific method of unbiased, measurable and reproducible inquiry with scrutiny by peer review as the best standard for evaluating the nature of illnesses and the effectiveness of medical diagnostic tests and therapies, and

BE IT FURTHER RESOLVED, that a physician who does not share this reliance cannot be an ethical practitioner of modern scientific medicine; and further declares that a physician who demonstrates that lack of reliance by habitually distorting, misrepresenting or selectively editing pertinent scientific data to patients is engaged in unethical and fraudulent conduct because of his/her failure to provide adequate informed consent to the patient.

RESOLUTION 90-09

SUBJECT: Lowering of Alaska's Presumptive Limit

WHEREAS, the epidemic of alcohol related motor vehicle accidents continues unabated leading to tens of thousands of deaths, hundreds of thousands of injuries, and millions of dollars in property damage annually, and

WHEREAS, demonstrated impairment of judgement at a blood alcohol level of 40 mg% has led the Federal Aviation Administration and the National Transportation Safety Board to establish that level as presumptive of intoxication for flying aircraft, and for operating other commercial vehicles including tankers, and

WHEREAS, the American Medical Association has embarked on a nationwide crusade to encourage states to lower their presumptive limits to 50 mg% from the usual 100 mg%, therefore

BE IT RESOLVED, that the Alaska State Medical Association seek the introduction of legislation to lower Alaska's presumptive limit to 50 mg% blood alcohol and/or 50 mg/210 liters of expired air.

RESOLUTION 90-11

SUBJECT: Physician Rotation Schedule with Magadan Hospital

WHEREAS, the era of peristroika has enhanced opportunities for physician interrelationships between the USSR and the USA; and

WHEREAS, Ted Mala, M.D. and the University of Alaska Institute for Circumpolar Health Studies have been instrumental in arranging opportunities for interchange in the north; and

WHEREAS, a new hospital is under construction in the Magadan area; and

WHEREAS, many physicians in Alaska have had experience in putting new hospitals into service; therefore

BE IT RESOLVED, that the Alaska State Medical Association express our appreciation to Dr. Mala and the University of Alaska Institute for Circumpolar Health for their continuing efforts in building international understanding; and

BE IT FURTHER RESOLVED, that the Alaska State Medical Association offer to help the physicians of Magadan in opening their new hospital, and should our offer be accepted

BE IT FURTHER RESOLVED, that a rotation schedule of Alaskan physicians in all specialties be established to provide ongoing assistance and cross-fertilization of ideas, experience, and knowledge.

RESOLUTION 90-12

SUBJECT: Request for Hearings by the Alaska State Medical Licensing Board

WHEREAS, there has been considerable public debate in Alaska as to the efficacy of certain unconventional medical therapies, and

WHEREAS, the public is understandably confused by the controversy, therefore

BE IT RESOLVED, that the Alaska State Medical Association requests that the Alaska State Medical Licensing Board, a public agency of the State of Alaska, convene a public hearing under the provisions of the Administrative Procedures Act (AS 44.62) to take testimony and arrive at a judgement as to whether certain practices constitute honest, competent and careful medical practice in the sense of the Medical Practice Statute (AS 08.64), particularly in relationship to the provisions of AS 08.64 (a) (2,3, and 8(A)). These practices in question include:

a. the use of chelation therapy for purposes other than treatment of heavy metal poisoning, reduction of excess blood calcium level, and/or digitalis overdosage; and

b. the recommendation to patients that the removal of dental amalgams will improve health and prevent mercury toxicity; and

c. the diagnosis and treatment of immune system deficiency other than as defined by the American Academy of Allergy and Immunology

RESOLUTION 90-13

SUBJECT: Condemning the Proposed Regulations Implementing the Clinical Laboratory Improvement Act of 1988

WHEREAS the proposed regulations for office laboratories published in the Federal Register, May 21, 1990 are exceedingly onerous, and

WHEREAS, their implementation would cause delay in outpatient diagnosis and treatment, and unnecessary expense in repetition of abnormal results, therefore

BE IT RESOLVED, that the Alaska State Medical Association ask in writing to HCFA and our congressional delegation that implementation of regulations implementing the Clinical Laboratory Improvement Act of 1988 be delayed until they can be revised.

RESOLUTION 90-15

SUBJECT: Endorsing the Health Access America Project

WHEREAS restricted access to affordable, high quality health care is a problem encountered by many Americans, including Alaskans; and

WHEREAS affordable insurance coverage and freedom of choice for appropriate health care services should be available to all Americans; and

WHEREAS the American Medical Association has developed Health Access America to ensure access by every citizen to the many benefits of the American health care system; therefore be it

RESOLVED that the Alaska State Medical Association endorses the principles set forth in Health Access America.

The elements of the AMA proposed plan may be summarized in the following 16 points:

1. Increase access by enacting major Medicaid Reform.
2. Increase access by requiring employer provision of health insurance.
3. Increase access by creating state-level risk pools in all states.
4. Maintain access and reduce costs for the elderly by enacting necessary Medicare Reform.
5. Increase access and reduce costs for the elderly by enacting necessary legislation to fi-

nance expanded long-term care coverage.

6. Reduce health care costs through professional liability reform.
7. Maintain quality and reduce costs through development of professional practice parameters.
8. Reduce health care costs through altering the tax treatment of employee health care benefits.
9. Reduce costs by encouraging cost-conscious decisions by patients.
10. Reduce costs by seeking innovation in insurance underwriting.
11. Maintain quality through expanded federal support for medical education, research and the National Institutes of Health (NIH).
12. Maintain quality and reduce costs through increased health promotion and disease prevention.
13. Reduce costs and increase access by amending ERISA or the federal tax code to equalize treatment of self-insured and insurance plans.
14. Reduce costs and increase access by repealing or overriding state-mandated benefit laws.
15. Reduce costs by reducing administrative costs and paperwork.
16. Maintain quality and access through encouraging physicians to practice in accordance with the highest ethical standards and to provide voluntary care.

RESOLUTION 90-16
SUBJECT: Supporting Appropriate Consumer Protection Legislation

WHEREAS, Alaska is one of a small number of states which has not enacted consumer protection legislation, and

WHEREAS, health fraud is a form of deceptive trade practice under consumer protection law, therefore

BE IT RESOLVED, that the Alaska State Medical Association supports and will actively participate in an effort to protect the Alaska public from health fraud as unfair and deceptive trade practices through advocacy of consumer protection legislation.

RESOLUTION 90-17
SUBJECT: Thanking Convention Committee, Staff, Speakers, Exhibitors and the people of Fairbanks

WHEREAS, a convention happens only as the considerable coordinated efforts of a host of folks, for the benefit of those who attend and the association as a whole, therefore

BE IT RESOLVED that the Alaska State Medical Association express our appreciation to:

1. The convention committee, for their planning and preparation;
2. The Alaska State Medical Association staff, for their tireless and friendly attention to the millions of little details and arrangements inherent in any convention;
3. The speakers, who stimulated us with new ideas and challenged us with their informed opinions;
4. The exhibitors, who financially undergird our meeting and bring worlds of products and services to assist us in caring for our patients; and
5. The people of Fairbanks, who have prepared our meals and lodging, shown us the local sights and welcomed us to their city.

RESOLUTION 90-18
SUBJECT: Reaffirmation of the Principles of Medical Ethics

BE IT RESOLVED, that the Alaska State Medical Association reaffirm our reliance on the American Medical Association Principles of Medical Ethics as guidelines to professional obligations and responsibilities, and reprints them here for information of our members; and

BE IT FURTHER RESOLVED, that the Alaska State Medical Association urge the State Medical Board to adopt these principles for dealing with Alaskan physicians.

PRINCIPLES OF MEDICAL ETHICS

PREAMBLE:

The medical profession has long subscribed to a body of ethical statements developed primarily for the benefit of the patient. As a member of this profession, a physician must recognize responsibility not only to patients, but also to society, to other health professions, and to self. The following Principles adopted by the American Medical Asssociation are not laws, but standards of conduct which define the essentials of honorable behavior for the physician.

I. A physician shall be dedicated to providing competent medical service with compassion and respect for human dignity.

II. A physician shall deal honestly with patients and colleagues, and strive to expose those physicians deficient in charcter or competence, or who engage in fraud or deception.

III. A physician shall respect the law and also recognize a responsiblity to seek changes in those requirements which are contrary to the best interests of the patient.

IV. A physician shall respect the rights of patients, of colleagues, and of other health professionals, and shall safeguard patient confidences within the constraints of the law.

V. A physician shall continue to study, apply and advance scientific knowledge, make relevant information available to patients, colleagues, and the public, obtain consultation, and use the talents of other health professionals when indicated.

VI. A physician shall, in the provision of appropriate patient care, except in emergencies, be free to choose whom to serve, with whom to associate, and the environment in which to provide medical services.

VII. A physician shall recognize a responsibility to participate in activities contributing to an improved community.

RESOLUTION 90-20
SUBJECT: Welcoming and Expressing our Appreciation to Governor Kobets, Dr. Lebedev and our Distinguished Colleagues Visiting from Magadan Region, USSR

WHEREAS, in the spirit of friendship and understanding Honorable Vachyeslav Kobets, Governor; and Dr. Alexsei Nikolaeovich Lebedev, Minister of Health of the Magadan Region, USSR have encouraged exchanges between their region and Alaska; and

WHEREAS, Dr. Aloa Kromaeva, obstetrician/gynecologist, Dr. Eduard Schubert, pathologist; and Dr. Hariton Bzgoed, surgeon, have dealt with paperwork and regulations, taken time from their duties at home, and made a long and arduous journey to this historic visit to our annual meeting; therefore

BE IT RESOLVED, that the Alaska State Medical Association thanks Governor Kobets, Dr. Lebedev, Dr. Kromaeva, Dr. Schubert and Dr. Bzgoed for their efforts in making this first-ever visit to our annual meeting possible; and

BE IT FURTHER RESOLVED, that the Alaska State Medical Association welcomes our visitors in the spirit of friendship and mutual respect, and in the interest of international understanding and exchange of medical knowledge; and

BE IT FURTHER RESOLVED, that the Alaska State Medical Association memorialize this historic visit by adopting as formal policy a program of increased exchange of medical knowledge and expertise with our colleagues in the Magadan Region, USSR.

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President's Corner

T'was my pleasure to attend the annual meeting of the American Medical Association in Chicago the last week in June. To see that organization in action was highly impressive and instilled pride in membership. The business of the association is conducted via resolutions which are submitted by state societies, individuals, or specialty groups. They are then reviewed by "reference committees", to which they are assigned on the basis of their general content. The committee reports to the delegates who then listen to floor debate and then vote the resolution up or down. The resolutions become policy which is carried out by staff under the direction of the board of trustees. I was greatly impressed by the brain power level of the delegates and the hard work exerted by them in evaluating the scores of resolutions with which they were required to become familiar. Our own Doug Smith and Dave Johnson were right in there with the best of them.

The inauguration of Dr. Tupper was a splendid affair, especially with the music of the Tommie Dorsey Band. The "Swing Era" band seems to be making a comeback - it's gratifying to a non-rock-N-roll fan like me to see many young kids in such outstanding bands. Maybe there's hope after all.

I appreciate the opportunity to attend as an alternate delegate. I became aware of a couple of small problems where I might be able to make a contribution. the resolutions will appear in due course.

Donald R. Rogers, M.D.
President
Alaska State Medical Association

NEW MEDICARE CLAIMS FILING REQUIREMENT

Recent Legislation

When Congress passed the Omnibus Budget Reconciliation Act of 1989, it included a requirement that all physicians and suppliers submit claims for Medicare beneficiaries beginning September 1, 1990. Congress believed that this would yield more accurate information with which to evaluate Medicare expenditures and other factors such as volume and intensity of services under the Medicare Volume Performance Standard (MVPS). The Standard is Congress's primary tool for managing the growth in Medicare Part B expenditures for physician services. For more information on the MVPS contact the Alaska State Medical Association.

Announcement

American Medical Association
Hospital Medical Staff Section
Sixteenth Assembly Meeting
November 29 - December 3, 1990

Medical Staffs from across the country are encouraged elect a medical staff representative to participate in the AMA-HMSS Assembly Meeting November 29 - December 1990 at The Peabody Orlando in Orlando, Florida.

The HMSS Assembly provides medical staffs with a unique opportunity to discuss and participate in the policymaking process of the AMA. In addition to the Assembly Meeting, an informative program on Economi Credentialing will be presented.

If you are unable to participate in the Orlando Meeting, we encourage you to call us with the name of your HMS Representative.

For further information about the AMA-HMSS, please call (312) 464-4754 or 464-4761.

EMS Symposium

The **Fourteenth Annual Alaska Symposium on Emergency** Medical Services will be held at the Hilton Hotel in Anchorage, Alaska on November 1-3, 1990. The symposium will provide continuing medical education credits applicable toward **EMT** and paramedic recertification requirements, **CEARP** points for nurses, and **Category 1** CME credits for physicians and physician assistants.
Contact: Karen Lee, Southern Region EMS Council, Inc., 6130 Tuttle Place, Anchorage, Alaska 99507 (907) 562-6449.

Auxiliary News

ALASKA STATE MEDICAL ASSOCIATION AUXILIARY

1989-90 was a fun-filled year for me as State President and my second year promises to be equally as satisfying. The State Auxiliary usually works in cooperation with the local auxiliary on health projects, community service programs etc. Those projects that are strictly state auxiliary programs are the scholarship program, AMA ERF Holiday Sharing Card fund raising, Alaska State Medical Association Auxiliary Convention, national health legislation projects.

One of the highlights of our convention in Fairbanks was the opportunity to visit with the Russian physicians and then to entertain them in Anchorage later. Some of your spouses were asked to take the Russian physicians into their offices or perhaps to the operating room for the day. Many of us had dinner parties and barbecues and now have wonderful stories to tell of our own "glasnost". It is my hope that we can continue to participate in the Alaskan Siberian connection through further affiliation with the Circumpolar Health Council. Ted Mala, M.D., Director, Institute for Circumpolar Health Studies, UAA, will be a guest lecturer at an upcoming state meeting.

One national health project that Alaska will participate in is a joint AMA ABA drug education format to be given in the class room to junior high school age children. A physician and attorney team will talk together on the medical and legal problems associated with drug use.

State auxiliary will also sponsor Organ Donor Awareness Week programs in April 1991. A planning workshop with guest speakers Kathy Andress, Organ Donation Coordinator and Steven Tucker, M.D. will be scheduled for January 1991. They will give us an overview of the donation process in the Pacific Northwest and how we can best educate the public about organ donation.

One last thing, at a convention program on Tort Reform we all heard how powerful auxiliaries across this nation can be. We have an opportunity to make some changes in Alaskan government right now, especially the Tort issue. Get involved, talk to your legislators and candidates, talk to your friends, raise your voice and make a difference.

ANCHORAGE MEDICAL SOCIETY

"The Great thing in this world is not so much where we are, but in what direction we are moving."
Oliver Wendell Holmes

Anchorage Medical Society Auxiliary - "Together Again"

A	ambitious and active members
U	undertakings, projects, and endeavors
X	[e]xamine ideas, values, purpose and goals
I	interest, enthusiasm, commitment
L	laughter, fun and togetherness
I	innovative and enjoyable speakers and programs
A	areas of participation: social, health, political
R	reaching out to others - friendship, love, and support
Y	year 1990-91 - A year to remember!

Put the Medical Auxiliary on your calendar this year.
It promises to be a good one! Let's move forward together.

Lorrie Horning, President
Fran Marbarger, Vice President
Gwen Hanley, Secretary
Jane Hall, Secretary
Gwen Geiringer, Treasurer

SCHOLARSHIP NEWS

The State Auxiliary Scholarship committee was delighted to award two scholarships last spring. The first recipient, Joe Roth is from Sitka and now a second year medical student at the University of Washington. Joe plans to practice medicine in rural Alaska upon completion of his residency. The second recipient is Nancy Morden from Haines, who will be a first year medical student at Harvard School of Medicine. Nancy has a special interest in public health. Both young people have varied backgrounds, including time as Peace Corps volunteers and international study programs abroad.

Lindsay Hansen Tucker
President
Alaska State Medical Association Auxiliary

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— Arrange manuscript as follows: (1) Title page including title, author(s), and location by city and state, institution at which work was done, address to which reprint requests should be sent. Authors will be sent three complimentary copies of the Journal in which their work appears. (2) Abstract of less than 150 words using no abbreviations, footnotes and references. (3) Text including introduction, methods, results and discussion. (4) Acknowledgements. (5) References, tables, figure legends and figures.

— References are cited in the text by numerals enclosed in parentheses. The reference section is typed double-spaced on sheets separate from the text and is numbered consecutively in the order in which references are cited in the text. Included are last names and initials of all authors, title of article, name of publication, volume, inclusive pages and year published. Abbreviations will conform to those used in **Index Medicus.**

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— Two complete sets of unmounted illustrations including photographs (black and white) should be submitted of uniform size not larger than 5x7 inches. These must be clearly labeled on the back, lightly in pencil, to indicate first author's name, figure number and top of illustration. Radiographs and photographs must be of high contrast.

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VASOTEC®

(ENALAPRIL MALEATE | MSD)

VASOTEC is available in 2.5-mg, 5-mg, 10-mg, and 20-mg tablet strengths.

Contraindications: VASOTEC® (Enalapril Maleate, MSD) is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor.

Warnings: *Angioedema.* Angioedema of the face, extremities, lips, tongue, glottis, and/or larynx has been reported in patients treated with ACE inhibitors, including VASOTEC. In such cases, VASOTEC should be promptly discontinued and the patient carefully observed until the swelling disappears In instances where swelling has been confined to the face and lips, the condition has generally resolved without treatment, although antihistamines have been useful in relieving symptoms. Angioedema associated with laryngeal edema may be fatal **Where there is involvement of the tongue, glottis, or larynx likely to cause airway obstruction, appropriate therapy, e.g., subcutaneous epinephrine solution 1:1000 (0.3 mL to 0.5 mL), should be promptly administered.** (See ADVERSE REACTIONS.)

Hypotension: Excessive hypotension is rare in uncomplicated hypertensive patients treated with VASOTEC alone Patients with heart failure given VASOTEC commonly have some reduction in blood pressure, especially with the first dose, but discontinuation of therapy for continuing symptomatic hypotension usually is not necessary when dosing instructions are followed; caution should be observed when initiating therapy (See DOSAGE AND ADMINISTRATION.) Patients at risk for excessive hypotension, sometimes associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death, include those with the following conditions or characteristics heart failure, hyponatremia, high-dose diuretic therapy, recent intensive diuresis or increase in diuretic dose, renal dialysis, or severe volume and/or salt depletion of any etiology. It may be advisable to eliminate the diuretic (except in patients with heart failure), reduce the diuretic dose, or increase salt intake cautiously before initiating therapy with VASOTEC in patients at risk for excessive hypotension who are able to tolerate such adjustments (See PRECAUTIONS, *Drug Interactions* and ADVERSE REACTIONS.) In patients at risk for excessive hypotension, therapy should be started under very close medical supervision and such patients should be followed closely for the first two weeks of treatment and whenever the dose of enalapril and/or diuretic is increased. Similar considerations may apply to patients with ischemic heart disease or cardiovascular disease in whom an excessive fall in blood pressure could result in a myocardial infarction or cerebrovascular accident If excessive hypotension occurs, the patient should be placed in the supine position and, if necessary, receive an intravenous infusion of normal saline. A transient hypotensive response is not a contraindication to further doses of VASOTEC, which usually can be given without difficulty once the blood pressure has stabilized If symptomatic hypotension develops, a dose reduction or discontinuation of VASOTEC or concomitant diuretic may be necessary

Neutropenia/Agranulocytosis: Another ACE inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression, rarely in uncomplicated patients but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease Available data from clinical trials of enalapril are insufficient to show that enalapril does not cause agranulocytosis at similar rates Foreign marketing experience has revealed several cases of neutropenia or agranulocytosis in which a causal relationship to enalapril cannot be excluded Periodic monitoring of white blood cell counts in patients with collagen vascular disease and renal disease should be considered

Precautions: *General. Impaired Renal Function* As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including VASOTEC, may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine were observed in 20% of patients These increases were almost always reversible upon discontinuation of enalapril and/or diuretic therapy In such patients, renal function should be monitored during the first few weeks of therapy.

Some patients with hypertension or heart failure with no apparent preexisting renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when VASOTEC has been given concomitantly with a diuretic. This is more likely to occur in patients with preexisting renal impairment Dosage reduction and/or discontinuation of the diuretic and/or VASOTEC may be required

Evaluation of patients with hypertension or heart failure should always include assessment of renal function. (See DOSAGE AND ADMINISTRATION.)

Hyperkalemia. Elevated serum potassium (>5.7 mEq/L) was observed in approximately 1% of hypertensive patients in clinical trials. In most cases these were isolated values which resolved despite continued therapy Hyperkalemia was a cause of discontinuation of therapy in 0.28% of hypertensive patients In clinical trials in heart failure, hyperkalemia was observed in 3.8% of patients, but was not a cause for discontinuation

Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with VASOTEC. (See *Drug Interactions*.)

Surgery/Anesthesia: In patients undergoing major surgery or during anesthesia with agents that produce hypotension, enalapril may block angiotensin II formation secondary to compensatory renin release If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion

Information for Patients.

Angioedema: Angioedema, including laryngeal edema, may occur especially following the first dose of enalapril Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to take no more drug until they have consulted with the prescribing physician

Hypotension: Patients should be cautioned to report lightheadedness, especially during the first few days of therapy If actual syncope occurs, the patients should be told to discontinue the drug until they have consulted with the prescribing physician

All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduction in fluid volume. Other causes of volume depletion such as vomiting or diarrhea may also lead to a fall in blood pressure; patients should be advised to consult with the physician

Hyperkalemia: Patients should be told not to use salt substitutes containing potassium without consulting their physician.

Neutropenia Patients should be told to report promptly any indication of infection (e.g., sore throat, fever) which may be a sign of neutropenia

NOTE As with many other drugs, certain advice to patients being treated with enalapril is warranted This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects

Drug Interactions:

Hypotension: Patients on Diuretic Therapy: Patients on diuretics and especially those in whom diuretic therapy was recently instituted may occasionally experience an excessive reduction of blood pressure after initiation of therapy with enalapril. The possibility of hypotensive effects with enalapril can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with enalapril. If it is necessary to continue the diuretic, provide close medical supervision after the initial dose for at least two hours and until blood pressure has stabilized for at least an additional hour (See WARNINGS and DOSAGE AND ADMINISTRATION.)

Agents Causing Renin Release: The antihypertensive effect of VASOTEC is augmented by antihypertensive agents that cause renin release (e.g., diuretics).

Other Cardiovascular Agents: VASOTEC has been used concomitantly with beta-adrenergic-blocking agents, methyldopa, nitrates, calcium-blocking agents, hydralazine, prazosin, and digoxin without evidence of clinically significant adverse interactions

Agents Increasing Serum Potassium. VASOTEC attenuates potassium loss caused by thiazide-type diuretics Potassium-sparing diuretics (e.g., spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium. Therefore, if concomitant use of these agents is indicated because of demonstrated hypokalemia, they should be used with caution and with frequent monitoring of serum potassium. Potassium-sparing agents should generally not be used in patients with heart failure receiving VASOTEC.

Lithium: Lithium toxicity has been reported in patients receiving lithium concomitantly with drugs which cause elimination of sodium, including ACE inhibitors A few cases of lithium toxicity have been reported in patients receiving concomitant VASOTEC and lithium and were reversible upon discontinuation of both drugs It is recommended that serum lithium levels be monitored frequently if enalapril is administered concomitantly with lithium.

Pregnancy–Category C: There was no fetotoxicity or teratogenicity in rats treated with up to 200 mg/kg/day of enalapril (333 times the maximum human dose) Fetotoxicity, expressed as a decrease in average fetal weight, occurred in rats given 1200 mg/kg/day of enalapril but did not occur when these animals were supplemented with saline Enalapril was not teratogenic in rabbits However, maternal and fetal toxicity occurred in some rabbits at doses of 1 mg/kg/day or more Saline supplementation prevented the maternal and fetal toxicity seen at doses of 3 and 10 mg/kg/day, but not at 30 mg/kg/day (50 times the maximum human dose).

Radioactivity was found to cross the placenta following administration of labeled enalapril to pregnant hamsters.

There are no adequate and well-controlled studies of enalapril in pregnant women. However, data are available that show enalapril crosses the human placenta Because the risk of fetal toxicity with the use of ACE inhibitors has not been clearly defined, VASOTEC® (Enalapril Maleate, MSD) should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Postmarketing experience with all ACE inhibitors thus far suggests the following with regard to pregnancy outcome Inadvertent exposure limited to the first trimester of pregnancy has not been reported to affect fetal outcome adversely Fetal exposure during the second and third trimesters of pregnancy has been associated with fetal and neonatal morbidity and mortality.

When ACE inhibitors are used during the later stages of pregnancy, there have been reports of hypotension and decreased renal perfusion in the newborn Oligohydramnios in the mother has also been reported, presumably representing decreased renal function in the fetus. Infants exposed *in utero* to ACE inhibitors should be closely observed for hypotension, oliguria, and hyperkalemia If oliguria occurs, attention should be directed toward support of blood pressure and renal perfusion with the administration of fluids and pressors as appropriate Problems associated with prematurity such as patent ductus arteriosus have occurred in association with maternal use of ACE inhibitors, but it is not clear whether they are related to ACE inhibition, maternal hypertension, or the underlying prematurity.

Nursing Mothers: Milk in lactating rats contains radioactivity following administration of ^{14}C enalapril maleate. It is not known whether this drug is secreted in human milk Because many drugs are secreted in human milk, caution should be exercised when VASOTEC is given to a nursing mother.

Pediatric Use Safety and effectiveness in children have not been established

Adverse Reactions: VASOTEC has been evaluated for safety in more than 10,000 patients, including over 1000 patients treated for one year or more VASOTEC has been found to be generally well tolerated in controlled clinical trials involving 2987 patients.

HYPERTENSION The most frequent clinical adverse experiences in controlled trials were: headache (5.2%), dizziness (4.3%), and fatigue (3%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in controlled clinical trials were: diarrhea (1.4%), nausea (1.4%), rash (1.4%), cough (1.3%), orthostatic effects (1.2%), and asthenia (1.1%).

HEART FAILURE The most frequent clinical adverse experiences in both controlled and uncontrolled trials were dizziness (7.9%), hypotension (6.7%), orthostatic effects (2.2%), syncope (2.2%), cough (2.2%), chest pain (2.1%), and diarrhea (2.1%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in both controlled and uncontrolled clinical trials were fatigue (1.8%), headache (1.8%), abdominal pain (1.6%), asthenia (1.6%), orthostatic hypotension (1.6%), vertigo (1.6%), angina pectoris (1.5%), nausea (1.3%), vomiting (1.3%), bronchitis (1.3%), dyspnea (1.3%), urinary tract infection (1.3%), rash (1.3%), and myocardial infarction (1.2%).

Other serious clinical adverse experiences occurring since the drug was marketed or adverse experiences occurring in 0.5% to 1% of patients with hypertension or heart failure in clinical trials in order of decreasing severity within each category

Cardiovascular Cardiac arrest, myocardial infarction or cerebrovascular accident, possibly secondary to excessive hypotension in high-risk patients (see WARNINGS, *Hypotension*); pulmonary embolism and infarction; pulmonary edema, rhythm disturbances, atrial fibrillation, palpitation.

Digestive: Ileus, pancreatitis, hepatitis (hepatocellular or cholestatic jaundice), melena, anorexia, dyspepsia, constipation, glossitis, stomatitis, dry mouth

Musculoskeletal Muscle cramps

Nervous/Psychiatric. Depression, confusion, ataxia, somnolence, insomnia, nervousness, paresthesia

Urogenital: Renal failure, oliguria, renal dysfunction (see PRECAUTIONS and DOSAGE AND ADMINISTRATION).

Respiratory Bronchospasm, rhinorrhea, sore throat and hoarseness, asthma, upper respiratory infection.

Skin: Exfoliative dermatitis, toxic epidermal necrolysis, Stevens-Johnson syndrome, herpes zoster, erythema multiforme, urticaria, pruritus, alopecia, flushing, hyperhidrosis

Special Senses Blurred vision, taste alteration, anosmia, tinnitus, conjunctivitis, dry eyes, tearing

A symptom complex has been reported which may include a positive ANA, an elevated erythrocyte sedimentation rate, arthralgias/arthritis, myalgias, fever, serositis, vasculitis, leukocytosis, eosinophilia, photosensitivity, rash, and other dermatologic manifestations.

Angioedema Angioedema has been reported in patients receiving VASOTEC (0.2%). Angioedema associated with laryngeal edema may be fatal If angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with VASOTEC should be discontinued and appropriate therapy instituted immediately (See WARNINGS.)

Hypotension. In the hypertensive patients, hypotension occurred in 0.9% and syncope occurred in 0.5% of patients following the initial dose or during extended therapy Hypotension or syncope was a cause for discontinuation of therapy in 0.1% of hypertensive patients. In heart failure patients, hypotension occurred in 6.7% and syncope occurred in 2.2% of patients Hypotension or syncope was a cause for discontinuation of therapy in 1.9% of patients with heart failure. (See WARNINGS.)

Clinical Laboratory Test Findings

Serum Electrolytes Hyperkalemia (see PRECAUTIONS), hyponatremia

Creatinine, Blood Urea Nitrogen: In controlled clinical trials, minor increases in blood urea nitrogen and serum creatinine, reversible upon discontinuation of therapy, were observed in about 0.2% of patients with essential hypertension treated with VASOTEC alone. Increases are more likely to occur in patients receiving concomitant diuretics or in patients with renal artery stenosis (See PRECAUTIONS.) In patients with heart failure who were also receiving diuretics with or without digitalis, increases in blood urea nitrogen or serum creatinine, usually reversible upon discontinuation of VASOTEC and/or other concomitant diuretic therapy, were observed in about 11% of patients Increases in blood urea nitrogen or creatinine were a cause for discontinuation in 1.2% of patients.

Hemoglobin and Hematocrit. Small decreases in hemoglobin and hematocrit (mean decreases of approximately 0.3 g% and 1.0 vol %, respectively) occur frequently in either hypertension or heart failure patients treated with VASOTEC but are rarely of clinical importance unless another cause of anemia coexists. In clinical trials, less than 0.1% of patients discontinued therapy due to anemia

Other (Causal Relationship Unknown) In marketing experience, rare cases of neutropenia, thrombocytopenia, and bone marrow depression have been reported A few cases of hemolysis have been reported in patients with G6PD deficiency

Liver Function Tests. Elevations of liver enzymes and/or serum bilirubin have occurred.

Dosage and Administration: *Hypertension:* In patients who are currently being treated with a diuretic, symptomatic hypotension occasionally may occur following the initial dose of VASOTEC The diuretic should, if possible, be discontinued for two to three days before beginning therapy with VASOTEC to reduce the likelihood of hypotension (See WARNINGS.) If the patient's blood pressure is not controlled with VASOTEC alone, diuretic therapy may be resumed

If the diuretic cannot be discontinued, an initial dose of 2.5 mg should be used under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour (See WARNINGS and PRECAUTIONS, *Drug Interactions*.)

The recommended initial dose in patients not on diuretics is 5 mg once a day Dosage should be adjusted according to blood pressure response. The usual dosage range is 10 to 40 mg per day administered in a single dose or in two divided doses. In some patients treated once daily, the antihypertensive effect may diminish toward the end of the dosing interval. In such patients, an increase in dosage or twice-daily administration should be considered If blood pressure is not controlled with VASOTEC alone, a diuretic may be added

Concomitant administration of VASOTEC with potassium supplements, potassium salt substitutes, or potassium-sparing diuretics may lead to increases of serum potassium (see PRECAUTIONS).

Dosage Adjustment in Hypertensive Patients with Renal Impairment: The usual dose of enalapril is recommended for patients with a creatinine clearance > 30 mL/min (serum creatinine of up to approximately 3 mg/dL). For patients with creatinine clearance ≤ 30 mL/min (serum creatinine ≥ 3 mg/dL), the first dose is 2.5 mg once daily The dosage may be titrated upward until blood pressure is controlled or to a maximum of 40 mg daily

Heart Failure. VASOTEC is indicated as adjunctive therapy with diuretics and digitalis The recommended starting dose is 2.5 mg once or twice daily. After the initial dose of VASOTEC, the patient should be observed under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions*.) If possible, the dose of the diuretic should be reduced, which may diminish the likelihood of hypotension. The appearance of hypotension after the initial dose of VASOTEC does not preclude subsequent careful dose titration with the drug, following effective management of the hypotension. The usual therapeutic dosing range for the treatment of heart failure is 5 to 20 mg daily given in two divided doses The maximum daily dose is 40 mg Once-daily dosing has been effective in a controlled study, but nearly all patients in this study were given 40 mg, the maximum recommended daily dose, and there has been much more experience with twice-daily dosing In addition, in a placebo-controlled study which demonstrated reduced mortality in patients with severe heart failure (NYHA Class IV), patients were treated with 2.5 to 40 mg per day of VASOTEC, almost always administered in two divided doses. (See CLINICAL PHARMACOLOGY, *Pharmacodynamics and Clinical Effects*.) Dosage may be adjusted depending upon clinical or hemodynamic response (See WARNINGS.)

Dosage Adjustment in Patients with Heart Failure and Renal Impairment or Hyponatremia: In patients with heart failure who have hyponatremia (serum sodium < 130 mEq/L) or with serum creatinine > 1.6 mg/dL, therapy should be initiated at 2.5 mg daily under close medical supervision. (See DOSAGE AND ADMINISTRATION, *Heart Failure*, WARNINGS, and PRECAUTIONS, *Drug Interactions*.) The dose may be increased to 2.5 mg b.i.d., then 5 mg b.i.d. and higher as needed, usually at intervals of four days or more, if at the time of dosage adjustment there is not excessive hypotension or significant deterioration of renal function. The maximum daily dose is 40 mg

For more detailed information, consult your MSD Representative or see Prescribing Information, Merck Sharp & Dohme, Division of Merck & Co., INC., West Point, PA 19486. J9VS61R2(819)

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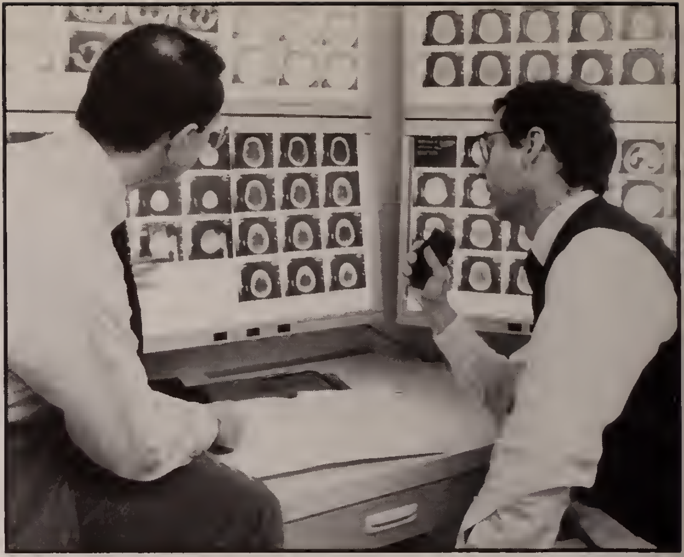
Quality Health Care In The Valley



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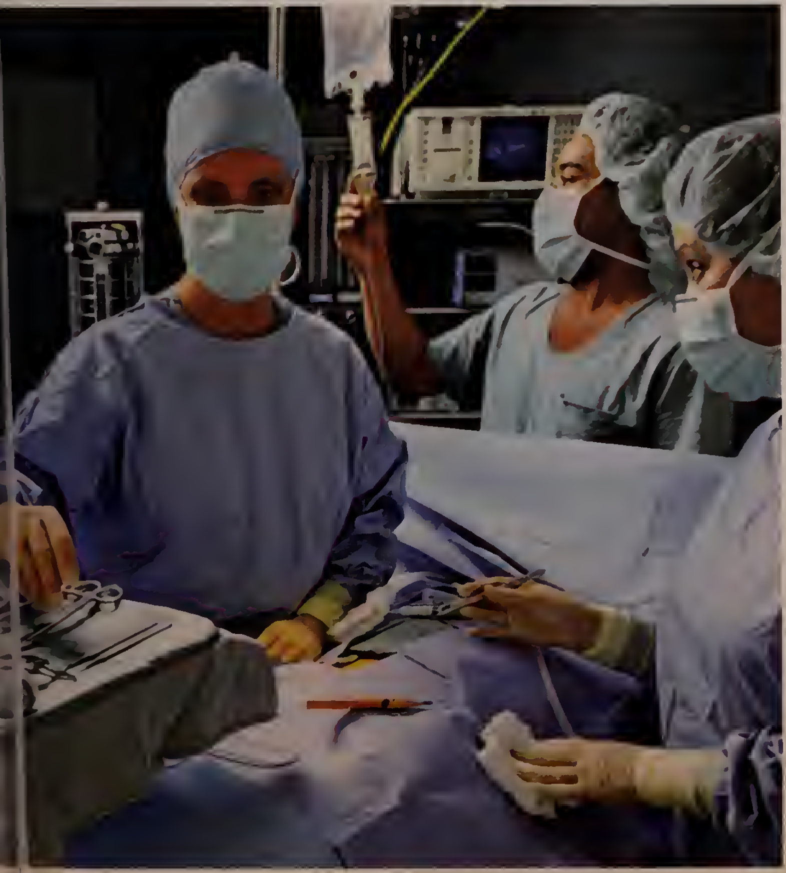
ARMY RESERVE MEDICAL PROFILE NO.9

Dr. Holwick outside of hospital where she practices as a civilian traumatologist.



Dr. Holwick in operating room at Letterman Army Medical Center.

JANN L. HOLWICK, M.D.

General and Trauma Surgeon.
Captain, U.S. Army Reserve.

EDUCATION University of Southern California, B.S.; University of California School of Medicine.

RESIDENCY Harbor General Hospital—UCLA Medical Center.

HOSPITAL AFFILIATIONS St. Luke Hospital; Huntington Memorial Hospital, Pasadena, California; Traumatologist, Arcadia Methodist Hospital, Arcadia, California.

OUTSTANDING ACHIEVEMENTS Borden Freshman Prize; Alpha Lambda Delta; Phi Beta Kappa; Phi Kappa Phi; Bovard Award; ALD Award; American Institute of Chemists Medal Award; Summa Cum Laude, University of California; Alpha Omega Alpha.

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Am Fam Phys 1987;36:133-140

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Brief Summary.
Consult the package literature for prescribing information.
Indication: Lower respiratory infections, including pneumonia, caused by *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Streptococcus pyogenes* (group A β-hemolytic streptococci).
Contraindication: Known allergy to cephalosporins.
Warnings: CECLOR SHOULD BE ADMINISTERED CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS. PENICILLINS AND CEPHALOSPORINS SHOW PARTIAL CROSS-ALLERGENICITY. POSSIBLE REACTIONS INCLUDE ANAPHYLAXIS.

Administer cautiously to allergic patients.

Pseudomembranous colitis has been reported with virtually all broad-spectrum antibiotics. It must be considered in differential diagnosis of antibiotic-associated diarrhea. Colon flora is altered by broad-spectrum antibiotic treatment, possibly resulting in antibiotic-associated colitis.

Precautions:

- Discontinue Ceclor in the event of allergic reactions to it.
- Prolonged use may result in overgrowth of nonsusceptible organisms.
- Positive direct Coombs' tests have been reported during treatment with cephalosporins.
- Ceclor should be administered with caution in the presence of markedly impaired renal function. Although dosage adjustments in moderate to severe renal impairment are usually not required, careful clinical observation and laboratory studies should be made.
- Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.
- Safety and effectiveness have not been determined in pregnancy, lactation, and infants less than one month old. Ceclor penetrates mother's milk. Exercise caution in prescribing for these patients.

Adverse Reactions: (percentage of patients)

Therapy-related adverse reactions are uncommon. Those reported include:

- Hypersensitivity reactions have been reported in about 1.5% of patients and include morbilliform eruptions (1 in 100). Pruritus, urticaria, and positive Coombs' tests each occur in less than 1 in 200 patients. Cases of **serum-sickness-like** reactions have been reported with the use of Ceclor. These are characterized by findings of erythema multiforme, rashes, and other skin manifestations accompanied by arthritis/arthralgia, with or without fever, and differ from classic serum sickness in that there is infrequently associated lymphadenopathy and proteinuria, no circulating immune complexes, and no evidence to date of sequelae of the reaction. While further investigation is ongoing, **serum-sickness-like** reactions appear to be due to hypersensitivity and more often occur during or following a second (or subsequent) course of therapy with Ceclor. Such reactions have been reported more frequently in children than in adults with an overall occurrence ranging from 1 in 200 (0.5%) in one focused trial to 2 in 8,346 (0.024%) in overall clinical trials (with an incidence in children in clinical trials of 0.055%) to 1 in 38,000 (0.003%) in spontaneous event reports. Signs and symptoms usually occur a few days after initiation of therapy and subside within a few days after cessation of therapy; occasionally these reactions have resulted in hospitalization, usually of short duration (median hospitalization = two to three days, based on postmarketing surveillance studies). In those requiring hospitalization, the symptoms have ranged from mild to severe at the time of admission with more of the severe reactions occurring in children. Antihistamines and glucocorticoids appear to enhance resolution of the signs and symptoms. No serious sequelae have been reported.
- Stevens-Johnson syndrome, toxic epidermal necrolysis, and anaphylaxis have been reported rarely. Anaphylaxis may be more common in patients with a history of penicillin allergy.
- Gastrointestinal (mostly diarrhea): 2.5%
- Symptoms of pseudomembranous colitis may appear either during or after antibiotic treatment.
- As with some penicillins and some other cephalosporins, transient hepatitis and cholestatic jaundice have been reported rarely.
- Rarely, reversible hyperactivity, nervousness, insomnia, confusion, hypertonia, dizziness, and somnolence have been reported.
- Other: eosinophilia, 2%; genital pruritus or vaginitis, less than 1% and, rarely, thrombocytopenia and reversible interstitial nephritis.

Abnormalities in laboratory results of uncertain etiology.

- Slight elevations in hepatic enzymes.
- Transient lymphocytosis, leukopenia, and, rarely, hemolytic anemia and reversible neutropenia.
- Rare reports of increased prothrombin time with or without clinical bleeding in patients receiving Ceclor and Coumadin concomitantly.
- Abnormal urinalysis; elevations in BUN or serum creatinine.
- Positive direct Coombs' test.
- False-positive tests for urinary glucose with Benedict's or Fehling's solution and Clinitest® tablets but not with Tes-Tape® (glucose enzymatic test strip, Lilly).

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Children receiving treats after medical treatment at a reindeer herders' camp, Magadan/Chukotka Region, U.S.S.R. Photo courtesy of Theodore Mala, M.D., M.P.H., Director, Institute for Circumpolar Health Studies, University of Alaska Anchorage.

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Infant Mortality in Alaska: Evidence of High Postneonatal Mortality Rate

Harry Harrison Jr., M.D.(1)
Anita Todd-Tigert, R.N.
Jack Jacob, M.D.

ABSTRACT

Improvements in infant mortality (death <1 year of age) have been experienced by the United States and Alaska over the past decade. The decline in the Alaska neonatal mortality (death <28 days of age) rate has been the major factor in our improved infant mortality rate. Alaska's neonatal mortality rate is one of the lowest in the U.S. However, postneonatal mortality (28 days to 1 year of age) rate has not declined and is one of the highest in the U.S. In this study we used vital statistics records to examine socio-demographic and inter-regional factors that may help to explain Alaska's apparently high postneonatal mortality rate. The study population consisted of all live born infants in Alaska for 1975 to 1985, who died in infancy. The Alaska neonatal mortality rate has been lower, but postneonatal mortality rate has been higher than the U.S. average for the period under consideration (p <.05). Comparison of Alaska postneonatal mortality rate from 1975 to 1985 with the U.S. found Alaska Non-natives have higher postneonatal mortality rates than U.S. White, and Alaska Native have higher postneonatal mortality rates than other U.S. minorities. The rural areas of Alaska have a higher postneonatal mortality rate than urban areas, a phenomenon also observed for the U.S. as a whole.

INTRODUCTION

Major improvements in infant mortality have been experienced by the United States and Alaska over the past decade. To a great extent, this can be attributed to the decline of the neonatal mortality rate resulting from the regionalization of perinatal care (7,8,13). While neonatal mortality reflects the outcome of prematurity and low birth weight births and adequacy of perinatal care access, postneonatal mortality is a more general indicator of the population's socio-economic status. To a lesser extent, the U.S. postneonatal mortality rate also declined. However, Alaska did not experience a decline in the postneonatal mortality rate during the same period. Analysis of postneonatal mortality (PNM) has traditionally involved examining factors such as regional geography, birth weight, ethnicity, income level, and maternal age and education. This descriptive investigation will compare the Alaska infant mortality statistics with the U.S. to illustrate the concern related to a high postneonatal mortality rate in Alaska. In addition, we will examine some of the major characteristics of postneonatal mortality.

METHODS

The study population consisted of all live born infants in Alaska, who died less than 1 year old. Neonatal mortality included the subset of infants who died at <28 days; postneonatal mortality included infants who died between 28 days and twelve months. The Alaska infant mortality rates were obtained from the National Center for Health Statistics (NCHS) monthly vital statistics report which was compiled by birth and death records from the state. The U.S. infant mortality rates were obtained from Wegman's Annual Infant Mortality Summary(15). The Alaska vital statistics section, Department of Public Health and Social Services, supplied ethnic-specific information from birth and death records. The period 1975 through 1985 was chosen because records were most complete during this time and they were calculated based on total number of annual infant deaths by total annual number of births (N/1000 births). Alaska census tract areas were grouped according to medical service areas or geographic proximity (table 1 and Alaska Map). The number of resident infants who may have died outside of Alaska was not determined.

The paired, two-tailed Student t-test was used in comparing infant, neonatal, postneonatal and ethnic-specific mortality rates. Based on the census tract area, mortality was compared using the Chi-square test. A linear regression analysis was used to determine trends of Alaska and the United States PNM rates. A relative risk was generated when comparing census tract-specific mortality rates using Anchorage as the referent. The Alaska relative risk for infant, neonatal and postneonatal mortality compared similar rates from the U.S. The relative risk is an indication of the magnitude of increased risk of mortality for a population. Factors related to postneonatal mortality, such as, birth weight and ethnicity, will be discussed in a subsequent study.

---

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Table 1.

1,2,3,4	N. Slope, Kobuk, Nome, Yukon (R)
5,6,7	Fairbanks, S.E. Fairbanks, W. Hampton (U)
8	Bethel (R)
9,11	Dillingham, Aleutians (R)
12	Matanuska-Suisitna Valley (U)
13	Anchorage (U)
14,15,16,17	Kenai Peninsula, Kodiak, Valdez, Skagway (U)
19,20,21	Juneau, Sitka, Wrangell, Price Wales,
22,23	and Ketchikan (U)

R=Rural

U=Urban and near Urban (direct Urban acccss)

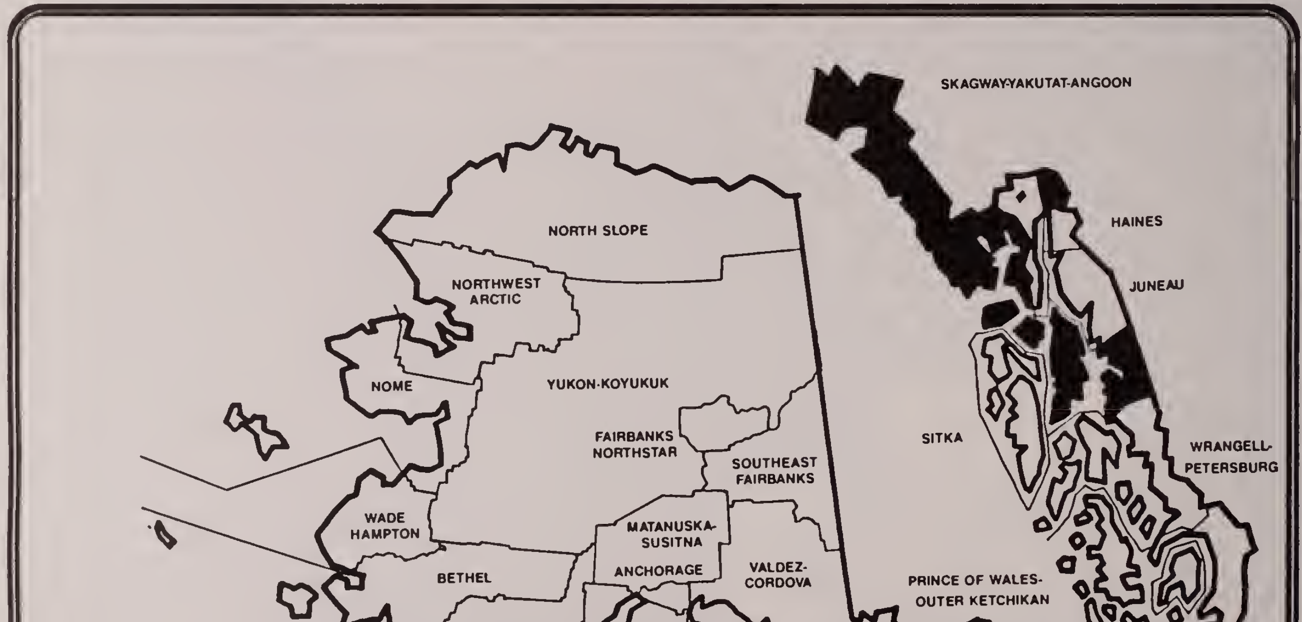
RESULTS

U.S. and Alaska infant mortality

During the eleven year period 1975 through 1985, the infant mortality rate in both the U.S. and Alaska progressively declined (figure 1). The U.S. infant mortality declined from 16/1000 in 1975 to 11/1000 in 1985, representing a relative reduction of 34%. The Alaska infant mortality declined from approximately 15/1000 in the late 1970s to approximately 12/1000 in the mid 1980s, representing a relative reduction of 20%. the mean infant mortality rate from 1975 to 1985 is 13.4% for the U.S. and 13% for Alaska. Because of such fluctuation related to small number, we employed simple linear regression to provide a best fit line for the Alaska data (annual infant mortality by year). The lines trend significant decline of Alaska infant mortality ($R^2=0.51$, $p=0.01$ (figure 1).

Neonatal mortality

Both the U.S. and Alaska neonatal mortality progressively declined during the past decade (figure 2). Since 1980, Alaska's neonatal mortal-



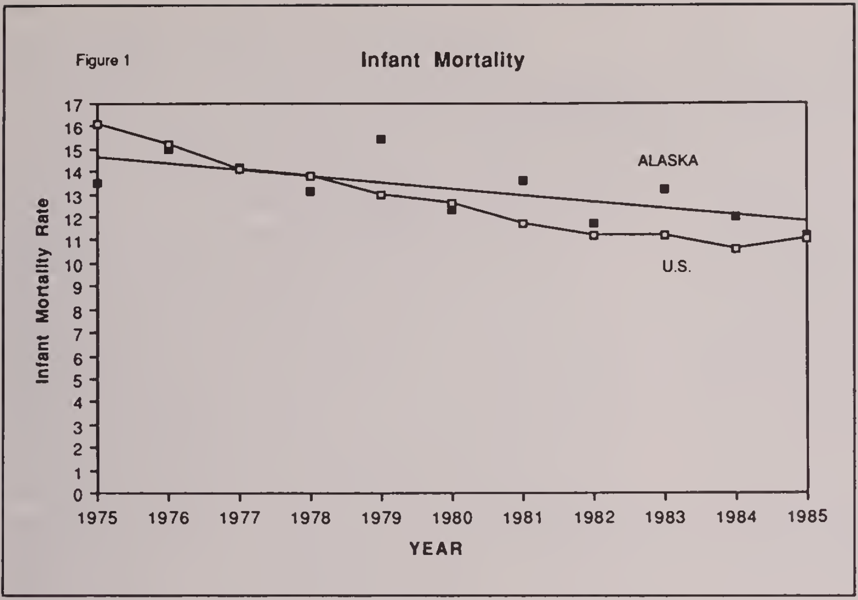


Figure 1: Infant mortality of Alaska and United States, 1975-1985.

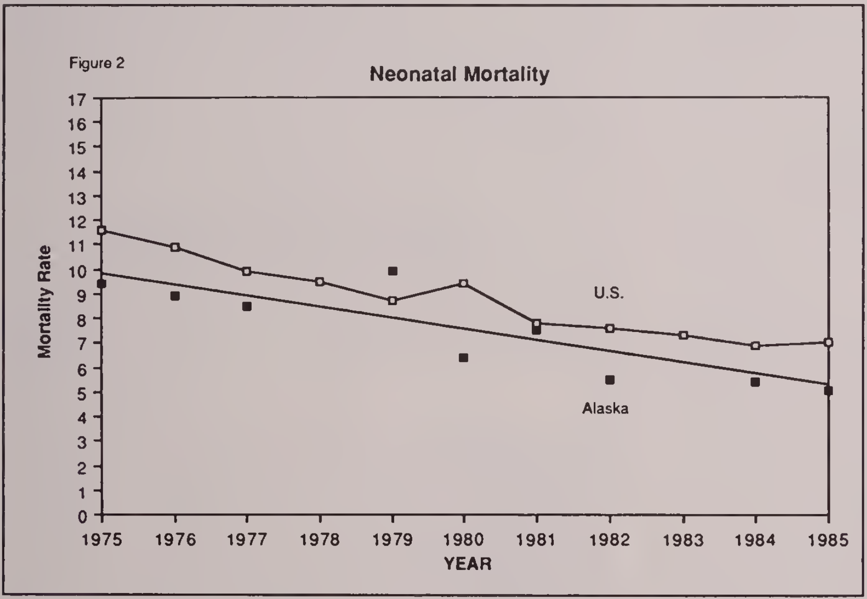


Figure 2: Neonatal mortality of Alaska and United States, 1975 - 1985.

ity rate has been equal to or less than that for the U.S. ($p < .05$) (8). The relative reduction of neonatal mortality (NM) for 1975 to 1985 is 39% for the U.S. and approximately 47% for Alaska.

Postneonatal mortality

The Alaska PNM rate has been higher than the U.S. average for the period under consideration (except for 1975 and 1978, figure 3.).

The U.S. PNM rate declined from 4.5/1000 to 3.7/1000 for 1975 to 1985 ($R^2=0.87$, $p<0.01$). However, Alaska PNM did not show a declining trend in the same time period ($R^2=0.28$, $p<0.1$). The United States ethnic-specific PNM rate progressively declined, although the Black PNM rate continues to be double the White PNM rate (Table 2). The Alaska PNM rate of 6.6 in 1984 exceeds that of the Black population of the United States. The three year (1983-1985) Alaska PNM rate demonstrates that the Native rate is 1.9 times higher than the non-Native rate ($p < 0.05$). the three year PNM rate for the Alaskan non-Native population is 36% higher than the U.S. White rate (p=NS), while the rate for Alaska Native is 35 percent higher when compared to the U.S. Black rate (p=NS). PNM should be a major concern of State health care planners and

Geographic variation of postneonatal mortality

The breakdown of Alaska by census area is seen in Table 3. These are the same census areas used by the State's Vital Statistic Section in 1986. Several census areas were grouped together by medical service area or geographic proximity. We used the census area (Anchorage) with the lowest PNM as the baseline to compare relative risk and showed that in most areas, except North Slope and Bethel, the relative risk is not statistically significant. Table 3 lists the PNM by region based on three year data (1983-1985). The two highest PNM areas are rural and predominantly Native. There were 215 total postneonatal deaths in 1983, 1984, and 1985. If the U.S. moving postneonatal mortality rate (3.8/1000) were applied to Alaska we would expect 142 deaths rather than the 215 observed ($X^2 < 0.05$).

DISCUSSION

The Alaska and U.S. infant mortality rates have shown a progressive decline over the past decade. From 1975 through 1985, neonatal mortality declined by 47 percent in Alaska. Since neonatal mortality constituted 3/4 of infant mortality in 1975 and was down to 1/2 of infant mortality in 1985, the 47 percent reduction of NM

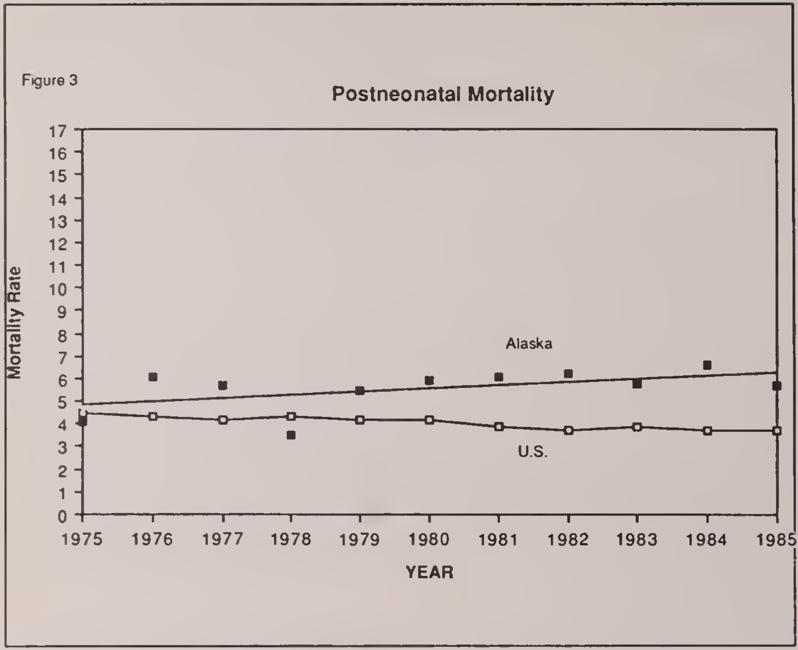


Figure 3: Postneonatal mortality of Alaska and United States, 1975-1985.

Table 2.

Comparison of Infant Mortality* United States and ALASKA

	1985	1984	1983
U.S.	10.6	10.8	11.2
Neonatal	7.0	6.9	7.3
Postneonatal	3.7	3.7	3.9
White	3.2	3.1	3.3
Black	6.1	6.5	6.8
Alaska	10.8	12.0	13.2
Neonatal	5.1	5.4	7.3
Postneonatal	5.7	6.6	5.8
Non-Native	4.0	4.5	4.8
Native	8.5	7.8	9.1

*Rate per 1000 live births.

accounts for most of the reduction during this period. To a great extent, the reduction of NM can be attributed to the efforts of regionalized perinatal care and tertiary center development which improved the survival of premature or low birth weight infants (3,4,11,12,13,14,16). Since 1980, Alaska has had one of the lowest neonatal mortality rates in the United States.

While the Alaska neonatal mortality rate is one of the lowest in the U.S. and continues to decline, the Alaska PNM rate is one of the highest in the U.S. with no evidence of decline. In contrast to the declining trend of U.S. PNM rate, the PNM rate gap between Alaska and U.S. appears to widen. This comparison of the PNM trend between Alaska and U.S. indicates that in recent years, Alaska had 1.8 times the PNM rate of the U.S. One potential explanation for the lack of progress of PNM rate decline for Alaska is related to limitation of statistics. The small number of births and deaths renders the PNM rate unstable compared to U.S. rate. Also, since the infant mortality rates reported by the NCHS are based on annual deaths and births instead of all matched births and deaths in a given year, some variation of rates can be introduced when annual number of births is changing as in the case in Alaska. However, the consistently higher PNM rate in Alaska indicates that the PNM rate is truly higher, and is unlikely to be accounted for by statistics or data quality. This trend of higher

Table 3.

Postneonatal Mortality by Census Area in Alaska for 1983-1985

Census Area	# of PNM	% of all PNM	PNM Rate by Region
N Slope (1-4)	23	10.7	9.4**
Fairbanks (5-7)	45	20.9	7.0
Bethel (8)	15	7.0	12.7**
Dillingham (9-11)	8	3.7	7.5
Matanuska-Susitna (12)	14	6.5	5.4
Anchorage (13)	68	31.6	4.3
Kenai Peninsula	21	9.8	4.9
Kodiak (14-17)			
Southeast (19-23)	21	9.8	5.7
Alaska Total	215		6.0*
U.S.			3.8

*Moving Average
**Signifies higher than State average

TDTH is total deaths

PNM Rate by Region per 1000 area live births.

PNM should be a major concern of State health care planners and providers (10,17).

One major factor for this discrepancy is the high PNM among Native infants which is 1.9 times higher than non=Native infants. Native births constitute approximately 1/3 of the births in the State, but account for 2/5 of the PNM. The North Slope and Bethel have significantly higher PNM rates than the rest of Alaska ($p<0.05$). These regions are the most remote with predominant Native populations. The known association of lower socio-economic status and increased risk of PNM would fit this pattern. However, other factors, such as residing in extreme rural environment with great distance to health care service may also play a role in the high rate of PNM. Native populations in the U.S. also have experienced a high PNM rate. Other factors, including postponement of neonatal mortality of low birth weight infants into the postneonatal period, due to increased survival, is the subject of another investigation (5,11).

A recent study indicated that Sudden Infant Death Syndrome (SIDS) is the leading cause of postneonatal death, especially among the Alaska Native population (1). However, Alaska does not have unified autopsy standards for investigating and evaluating infants who may die of SIDS, so this etiologic category may contain a subset of infants dying of other causes. Review of many autopsy reports during the last four years has disclosed major variations from autopsy standards suggested by Beckwith and Keeling (2,9). The issue of the contribution of SIDS or other diseases simulating SIDS as the cause of high PNM in Alaska also requires further investigation.

The geographic dimensions and rural nature of the State also raises problems of adequate medical care access (6). This is of particular importance when considering tertiary pediatric intensive care. Major cases of postneonatal morbidity, infection, major and minor congenital anomalies, and accidents, may not gain access to specialized medical care until the infant is severely compromised owing to the distance involved in a medical evacuation. Patterns of medical care access are under investigation.

Perhaps the high PNM problem in Alaska could be better defined by conducting detailed characterization of all PNM cases through a review process. The review mechanism might identify potential sources of improvement in health care delivery which cannot be uncovered by vital record analysis.

ACKNOWLEDGMENT

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Plasmapheresis: First Year of Experience at Humana Hospital-Alaska

by Richard L. Neubauer, M.D.⁽¹⁾
Gerald G. Morris, M.D.

SUMMARY

This report summarizes the first year of experience with therapeutic plasmapheresis at Humana Hospital-Alaska. Eleven patients had plasmapheresis; 6 patients with the Guillain-Barré syndrome, and patients with thrombotic thrombocytopenic purpura (TTP), Eaton-Lambert syndrome, myasthenia gravis, post transfusion purpura, and multiple myeloma complicated by hyperviscosity and coagulopathy. Many of our patients appeared to benefit from plasmapheresis, but we also report a variety of complications. Our approach to establishing plasmapheresis in a community hospital setting is discussed.

INTRODUCTION

Therapeutic plasmapheresis has been performed in a wide variety of illnesses, but the indication for plasmapheresis are not always clear-cut. It has been suggested that plasmapheresis only be used in diseases where a rationale for use is firmly grounded in theoretical terms or where a definite clinical utility has been demonstrated (1). This situation is confounded by the fact that many of the illnesses for which plasmapheresis has been performed have a high fatality rate and are also rare. Thus it is unlikely that a controlled trial will ever be done to study the efficacy of plasmapheresis in treating many of these diseases.

In early 1988, at the urging of the medical staff, Humana Hospital-Alaska put in service a plasmapheresis unit. We report here our first year of experience with therapeutic plasmapheresis. It is noteworthy that in establishing the plasmapheresis program, the department of Internal Medicine issued guidelines that were aimed at ensuring the use of this technology for disease processes where the utility is reasonably well established. The guidelines do not restrict the use of plasmapheresis to a specified list of conditions, but experimental uses such as the treatment of multiple sclerosis or amyotrophic lateral sclerosis were not felt appropriate to our community hospital setting.

DEMOGRAPHICS

The demographics of our patient population is outlined in Table 1. The patients range in age from 13 to 74, and the sex distribution was roughly equal male to female. Intravenous access for plasmapheresis was via peripheral veins in four of the patients, and via an A-V fistula in one patient who had underlying chronic renal failure. In the others, central access was used. These patients received plasmapheresis between April 1988 and September 1989.

Table 1.
Plasmapheresis
Humana Hospital-Alaska 1988-1989

Number of Patients	11
Ages (years)	
range	13-74
mean	42
Sex	6 male, 5 female

Diseases Treated with Plasmapheresis

Table 2 outlines the conditions for which our patients underwent plasmapheresis. The Guillain- Barré syndrome has become a well accepted indication for plasmapheresis (2-6). The majority of our patients (6/11) received plasmapheresis treatments for the Guil-

Table 2.

Diseases Treated with Plasmapheresis

Disease Entity	Number of Patients
Guillain-Barré	6
Eaton-Lambert	1
Myasthenia Gravis	1
TTP	1
Post Transfusion Purpura	1
Hyperviscosity & Coagulopathy (In Multiple Myeloma)	1

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lain-Barré syndrome. One patient each had TTP, Eaton-Lambert syndrome, myasthenia gravis, post transfusion purpura, and multiple myeloma complicated by hyperviscosity and coagulopathy.

Outcome of Plasmapheresis

While no conclusions regarding the efficacy of plasmapheresis can be drawn from such a small sample of patients with a wide variety of disease entities, several of our patients are worthy of special mention.

The first patient to undergo plasmapheresis in our institution has already been reported (7). She had TTP, did not respond to infusion of fresh frozen plasma, and her recovery was temporally related to plasmapheresis. She had a relapse which was again successfully treated with plasmapheresis, and now remains well two years after her initial illness.

Two of the six patients with Guillain-Barré syndrome had dramatic recoveries that may be related to the use of plasmapheresis. Both of these patients were intubated and on ventilators, and had prognostic indicators that pointed to the potential need for prolonged ventilatory support. Plasmapheresis was initiated early in the course of the illness, and they were weaned from ventilatory support within days. While the rest of the patients with Guillain-Barré syndrome responded less dramatically, they also appeared to improve when plasmapheresis was instituted.

The patient with post transfusion purpura was very ill with cardiorespiratory failure due to coronary artery disease and valvular heart disease. She developed post transfusion purpura related to transfusions for hemorrhagic gastritis. The etiology of post transfusion purpura is felt to be linked to alloimmunization against PLA[1] platelet antigen, although there remain some significant questions regarding pathogenesis of this rare condition (8-11). Reported treatment modalities for post transfusion purpura have included infusion of gamma globulin, corticosteroids, and plasmapheresis (12-16).

This patient was plasmapheresed at a time when her platelet count was zero, and there was a significant rise in the platelet count coincident with the initiation of plasmapheresis. Plasmapheresis was initiated in this patient only after more conservative treatment with intravenous gamma globulin infusions and corticosteroids had failed.

There was one death that was temporally related to plasmapheresis. A 74-year-old man with Guillain-Barré had a cardiac arrest, characterized by electromechanical dissociation, shortly after a plasmapheresis session. At autopsy, he was found to have coronary artery disease, and evidence of a recent myocardial infarction. There was no evidence of complications from the central catheter via which the plasmapheresis had been performed. Our conclusion is that this death was unrelated to plasmapheresis.

Complications of Plasmapheresis

Table 3 outlines the complications that were clearly related to plasmapheresis in these 11 patients. It is evident that plasmapheresis in our patient population was associated with a significant number of complications that are generally of the type that would be expected with this procedure.

Table 3.

Complications of Plasmapheresis

Complications	Number of Patients
Catheter Sepsis	1
Urticaria	1
Thrombosis of AV Fistula	1
Acute Hepatitis/Etiology	1
Non-A, Non-B Hepatitis	1
Exacerbation of CHF	1
Hypotension	1

Several of these complications are worthy of special mention. Catheter sepsis occurred on multiple occasions in our 13 year-old patient with myasthenia gravis. She had three successive Hickman catheters placed, and it was suspected but not proven that purposeful contamination of the catheters might be a factor. She was lost to follow up when she left Alaska.

One patient with Guillain-Barré syndrome developed an acute hepatitis after several plasmapheresis sessions. This patient received only 5 per cent albumin replacement fluid during pheresis, and the rise in hepatic enzymes occurred over a period of hours after plasmapheresis. This patient also had a recent kidney transplant for chronic renal failure, was receiving immunosuppressive drugs, and it was unclear whether the hepatitis was related to the plasmapheresis.

Non-A, non-B hepatitis developed in the patient with TTP. This patient received over 100 units of fresh frozen plasma over the course of 13 plasmapheresis sessions. As reviewed in the previous report on this patient, fresh frozen plasma must be used in plasmapheresis for TTP, despite the risk of exposure to blood borne viruses (7). She recovered uneventfully from the hepatitis.

The patient with post transfusion purpura had severe congestive heart failure at the time of her first plasmapheresis, and worsened at the end of the first session. This was treated with aggressive medical therapy. During a subsequent session, an attempt was made to remove volume during plasmapheresis, and the patient became hypotensive. This responded to a fluid bolus.

In the patients from this series every effort was made to use five per cent albumin as a replacement fluid, since it does not carry a known risk of transmitting blood borne viruses. As previously discussed, fresh frozen plasma must be used as a replacement fluid in patients with TTP, and modest amounts of fresh frozen plasma are also used when the fibrinogen level falls during repeated plasmapheresis sessions. The fall in fibrinogen level can often be minimized by performing plasmapheresis on every other day rather than daily. It is noteworthy that transmission of the AIDS virus has been reported from plasmapheresis using fresh frozen plasma(17).

CONCLUSION

Plasmapheresis is a controversial technology. Because of our remote location, the medical community urged that this capability be made available despite the recognition that plasmapheresis has relatively few indications, generally for very rare diseases.

From the outset, we were determined to restrict use of this technology to illnesses where reasonably good evidence supported its utility.

As can be seen from the complications that we report, plasmapheresis is not a totally benign technique. We will continue to monitor the use of plasmapheresis, and believe that it has added a useful therapeutic strategy for selected indications.

ACKNOWLEDGMENT

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HEART DAY

AN EXPERIENCE IN CARDIOVASCULAR RISK FACTOR DETECTION IN A RURAL ALASKAN POPULATION.

R.O. Van Camp, M.D.(1)

ABSTRACT

This is a summary of the results of a project to influence cardiovascular risk factors in a rural population using a community based clinic with very limited financial and staffing resources. The project, which was well received by the community, was successful in influencing community-wide awareness of cardiovascular risk factors and in screening many people who would otherwise not receive medical care. A significant number of correctable risk factors for cardiovascular disease were discovered.

INTRODUCTION

Glennallen is a small unincorporated town of approximately 400 people. It lies in the Copper River Valley, at the intersection of two highways in southcentral Alaska. A small clinic, Cross Road Medical Center, is the only physician health care provided in the area. It incorporates a lab, pharmacy, clinic, emergency department and holding facility. Most of the 3500 residents of the area live along the road system, within 60 miles of the clinic. The Heart Day project was planned to promote wellness, detect risk factors for cardiovascular disease, and give immediate feedback on how these risks can be decreased.

METHODS

A publicity program was organized which included newspaper, radio and posters. The information was creatively packaged and followed the public education guidelines developed by the American Heart Association. A Saturday was chosen to encourage participation. Blood tests for cholesterol and glucose were done accurately, quickly and at low cost.

People were enrolled, then immediately taken to the laboratory. Blood was usually drawn by venipuncture, however a capillary method was available for those who declined venipuncture. Plasma cholesterol and glucose levels were performed on a Kodak DT-60. Height, weight and blood pressure measurements were taken by the nurses while blood was processed by our Laboratory Technologist. Family history pertaining to cardiovascular disease was taken. History of symptoms of cardiovascular disease, smoking and alcohol use were determined. A nurse or physician gave short teaching sessions and pamphlets to reinforce healthy life styles and encourage steps to eliminate any correctable risk factors. This was usually accomplished in less than an hour of the participant's time.

RESULTS

One hundred thirty-five people were tested in the seven hour period allowed. See Table 1 for a summary of the data and Table 2 for the complete data. A wide section of the population was represented. Equal numbers of men and women were tested and the age distribution was fairly representative of the total population. Important correctable risk factors were determined in 75 per cent of the people evaluated. Approximately two thirds of the participants had a family history suggesting that they might be at an increased risk for heart disease. Equal numbers of men and women used tobacco or alcohol. Eight per cent of the adults tested admitted tobacco use, and 25 per cent admitted alcohol use. No one admitted smoking more than one pack per day. Measurements showed that: 56 per cent had cholesterol levels of 200mg/dl or greater; 16 per cent had systolic blood pressures of 140mm of Hg or higher; and 45 per cent were more than 20 pounds overweight. Carbon Monoxide levels were performed on the venous specimens with no significant poisoning detected.

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(1)Cross Road Medical Center, Glennallen, Alaska

Some people who needed prompt medical attention were identified. Six had systolic blood pressures greater than 160mm of Hg and one person who was not symptomatic enough to seek any medical attention had a blood glucose of 333 mg/dl.

The cost of the screening was offset by the ten dollar charge for the blood tests. Eighty percent of the clinic staff which included six nurses, the lab tech, physician and administrator volunteered their time. Extra demands were placed on a staff that was already tired from multiple medical emergencies occurring over the previous two days and nights.

CONCLUSION

Public health screening for risk factors of heart disease can be done in a rural area with good response from the community. The experience can be rewarding for the medical staff involved but unwelcome stress can occur. A larger than expected proportion of the responders had significant family or past medical history that would place them at higher risk for heart disease. A low but significant percentage of people receive immediate benefit. Long term benefits are expected as new committments to health are made.

ACKNOWLEDGEMENTS:

Appreciation is expressed to the entire administrative and medical staff of Cross Road Medical Center who worked hard to make this project a success. A special thanks to Sharlane Donaldson, RT who tabulated most of the data and was responsible for much of the organization.

Table 1. Summary of Risk Factor Findings

AGE by decade	PARTICIPANTS *total (male)	SYSTOLIC BP 140 mmHg	CHOLESTEROL >200 mg/dl	OBESE	SMOKERS	ETOH use	+FAMILY HISTORY
0-10	5 (5)	0	0	0	0	0	5 (5)
11-20	7 (3)	0	0	2 (1)	0	0	6 (2)
21-30	6 (3)	0	2 (1)	3 (1)	1 (l)	5 (3)	4 (2)
31-40	46 (18)	2 (2)	23 (11)	18(5)	5 (2)	10 (6)	31 (11)
41-50	34 (17)	4 (1)	24 (11)	17(8)	1 (1)	13 (6)	26 (12)
51-60	13 (6)	5 (2)	11 (4)	12(6)	2 (1)	3 (1)	10 (5)
61-70	18 (8)	7 (2)	12 (5)	8 (4)	1 (0)	3 (2)	13 (6)
71+	6 (3)	4 (2)	4 (2)	3 (2)	1 (1)	0	0
TOTAL	135 (63)	22 (9)	76 (34)	63 (27)	11 (6)	34 (18)	95 (43)
%TOTAL	100 (47)	16 (14)	56 (53)	46 (42)	8 (9)	25 (28)	70 (68)

NOTES:

Totals unparenthesized, males in parentheses

Obese=over 20% above ideal weight

+Family history=Patient reports history of elevated cholesterol, stroke, hypertension or heart attack in family.

Table 2. Summary of All Data

Pt#	AGE	SEX	CHOLEST mg/dl	SYS BP mm Hg	DIAS BP mm Hg	GLUCOSE mg/dl	WT Cat.	SMOKES pk/day	ETOH oz/wk	FH	SX
613	6	M	179	96	60	61	A	0	o	+	o
588	7	M	144	88	62	88	A	0	o	+	o
612	8	M	170	100	68	77	A	0	o	+	o
636	10	M	113	90	60	68	A	0	o	+	o
637	10	M	141	110	70	81	A	0	o	+	o
537	11	F	144	110	68	106	A	0	o	+	o
587	12	M	122	116	68	87	A	0	o	+	o
663	12	M	146	128	70	122	C	0	o	+	o
538	13	M	145	120	80	105	A	0	o	+	o
615	17	F	160	110	60	86	B	0	o	+	o
586	17	F	133	136	68	93	C	0	o	+	o
608	20	F	193	114	80	104	B	0	o	+	o
606	23	F	230	120	80	77	D	0	o	+	o
582	24	F	194	122	80	77	D	0	1	+	o
661	26	M	193	116	80	82	C	0	2	+	o
599	26	M	175	112	52	100	B	1	10	+	o
559	27	M	238	116	78	81	B	0	1	o	o
546	30	F	183	120	70	86	A	0	2	+	o
532	31	F	188	86	60	97	A	0	o	o	o
548	31	M	145	124	80	71	B	0	o	+	o
649	31	M	154	108	68	90	C	0	o	+	o
566	32	M	247	112	60	95	B	0	o	+	o
651	32	F	226	118	82	84	B	0	o	+	+
539	33	F	198	120	82	95	B	0	o	+	o
633	33	M	187	116	80	78	C	1/2	2	o	o
618	34	F	170	108	70	79	A	1/2	+	+	o
619	34	M	206	120	80	109	B	1	o	+	o
653	34	M	129	118	86	86	B	0	o	o	o
640	34	F	183	126	90	80	C	1/2	o	+	o
604	34	F	187	102	62	87	B	0	7	+	o
662	35	F	232	110	70	88	D	0	o	o	o
652	35	F	200	102	70	88	C	0	o	+	o
644	35	F	232	134	84	117	A	0	o	+	o
544	35	F	149	122	78	95	D	0	1	+	o
601	35	F	171	110	60	83	C	0	o	o	o
547	35	M	239	144	98	86	B	0	14	o	o
564	35	M	217	118	78	94	C	0	0	+	o
650	36	F	231	120	80	66	B	0	o	+	o
631	36	M	200	132	90	87	C	0	o	o	o
545	36	M	165	138	84	72	B	0	2	+	+
540	36	F	212	132	82	111	B	0	o	+	o
565	36	M	187	142	86	96	A	0	o	o	o
614	37	F	188	88	58	84	D	0	o	+	o
535	37	F	134	112	70	102	B	0	o	+	o
605	37	M	172	120	80	82	B	0	7	+	o
577	37	F	233	118	80	94	B	1	o	o	o
632	37	F	147	104	70	78	B	0	o	+	o
623	37	F	118	110	80	93	C	0	o	o	o
635	37	F	161	112	80	73	B	0	o	o	o
530	37	F	240	104	68	99	C	0	o	+	o
536	38	M	262	138	100	102	B	0	o	+	o
571	38	F	133	118	68	76	B	0	4	o	o
628	38	F	223	100	70	84	C	0	o	+	o
634	39	M	233	122	90	83	B	0	o	+	o
603	39	M	196	102	78	90	C	0	7	+	o
552	39	F	187	118	78	83	C	0	o	+	o
602	39	M	277	102	60	91	B	0	o	+	o
660	39	F	215	114	80	88	D	0	o	+	o
542	40	F	226	112	68	86	B	0	o	o	o
595	40	F	185	104	60	85	B	0	o	+	o
531	40	M	212	120	80	99	B	0	+	+	o

Table 2. continued

563	40	F	269	126	94	93	D	0	o	+	o
611	40	M	203	124	84	84	A	0	o	+	+
625	40	F	255	114	60	97	C	0	o	+	+
572	41	M	201	122	80	91	C	0	o	+	o
648	41	F	179	112	80	106	B	0	o	+	o
573	41	F	173	112	72	92	A	0	o	+	o
645	42	M	152	100	80	106	B	1	2	o	o
585	42	F	186	140	90	81	C	0	o	o	o
590	43	F	201	110	74	85	C	0	o	+	+
567	43	M	178	122	70	87	B	0	o	o	o
646	44	M	227	116	80	87	B	0	o	o	o
560	44	F	211	120	78	103	B	0	+	o	o
624	44	M	232	124	90	105	C	0	o	+	o
551	44	M	258	128	98	98	C	0	o	o	o
655	44	F	246	116	80	136	D	0	3	+	o
622	44	M	181	124	70	90	B	0	o	+	o
630	44	F	171	128	80	99	D	0	o	+	o
658	45	F	203	120	70	91	B	0	1	+	o
626	45	F	229	132	90	92	D	0	o	+	+
607	45	F	208	108	76	81	B	0	o	+	o
591	45	M	218	122	80	100	D	0	o	+	o
553	45	F	229	124	90	83	B	0	2	+	o
570	45	M	210	140	80	97	C	0	5	+	o
659	46	M	213	128	80	88	D	0	3	+	o
638	46	M	248	104	70	83	B	0	o	+	o
534	47	F	261	112	82	112	B	?	2	o	o
639	49	M	197	138	100	92	C	0	2	+	o
629	49	M	233	124	90	104	B	0	4	o	o
578	49	F	214	112	70	87	B	0	o	+	o
627	49	M	220	112	80	92	A	0	o	+	o
575	49	M	249	138	80	96	D	0	+	+	o
579	49	M	187	90	70	71	B	0	o	+	o
554	49	M	197	118	72	86	A	0	o	+	o
533	49	F	235	138	92	116	C	0	+	+	0
589	49	F	269	160	94	92	D	0	o	+	o
584	49	F	269	120	86	94	C	0	+	+	o
576	49	F	219	140	78	106	D	0	o	+	o
654	51	F	239	144	84	92	D	0	o	+	o
556	52	M	252	120	82	94	C	0	o	o	o
610	54	F	242	150	86	84	B	0	o	+	+
583	55	F	200	120	80	104	C	0	+	o	o
574	56	F	238	188	92	91	D	1	+	+	o
561	56	M	232	122	88	88	C	1/2	+	+	o
562	57	F	285	128	80	91	C	1	o	o	o
641	57	M	174	112	80	103	D	0	o	+	o
617	58	F	212	100	68	105	C	0	o	+	o
569	58	M	219	142	80	85	C	0	o	+	o
656	58	M	258	150	96	102	C	0	o	+	o
557	59	F	264	120	80	101	C	0	o	+	o
558	60	M	156	136	100	175	C	0	o	+	o
609	61	M	191	110	68	95	B	0	7	+	o
581	61	F	204	178	94	95	C	0	o	o	o
592	61	F	233	118	80	98	D	0	o	+	o
580	61	M	261	110	80	95	C	0	o	+	o
549	62	F	263	180	110	100	D	0	2	+	o
616	63	F	185	80	50	93	B	0	o	o	o
598	63	M	196	120	70	87	B	0	o	+	o
555	63	F	261	160	82	333	B	0	o	+	o
597	63	F	213	122	70	65	B	0	o	+	+
568	64	F	251	148	72	82	B	0	o	+	o
593	64	M	134	134	90	82	A	0	o	+	o
543	65	F	198	130	80	90	B	0	o	o	+
550	66	M	245	150	88	86	C	0	2	o	o
657	66	M	233	156	88	105	D	0	o	o	o
621	67	M	201	114	70	76	C	0	o	+	o
596	68	F	234	136	80	96	D	0	o	+	o

Table 2. Continued

600	69	F	194	142	64	77	A	1/2	o	+	o
541	70	M	217	128	100	104	B	0	o	+	o
642	73	F	277	108	60	118	B	0	o	o	o
594	75	M	163	172	84	101	C	1	o	o	o
642	73	M	209	148	80	105	A	0	o	o	+
528	78	F	260	146	64	120	D	0	o	?	+
529	78	M	250	92	68	103	C	0	o	?	o
620	80	F	170	190	100	69	B	0	o	o	o

Notes:

Weight Categories:	A=ideal range	C=121-140%
	B100 - 120% of ideal	D=over 141%

Family History= A family history was considered positive if a person reported a near relative with high cholesterol, "high blood pressure," a "stroke," or a "heart attack."

Symptoms=A person was considered symptomatic if they reported chest pain that had prompted a cardiac evaluation, OR would probably have done so, had they sought medical assistance.

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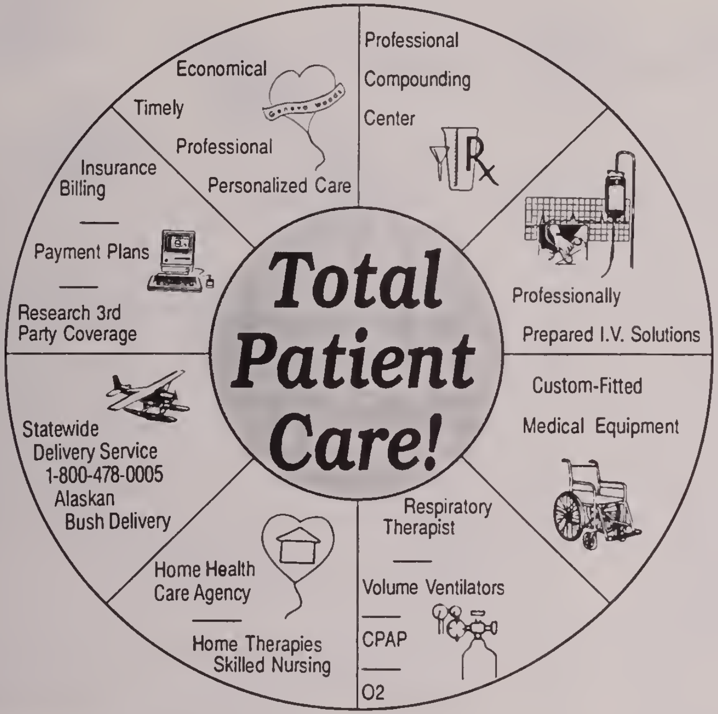
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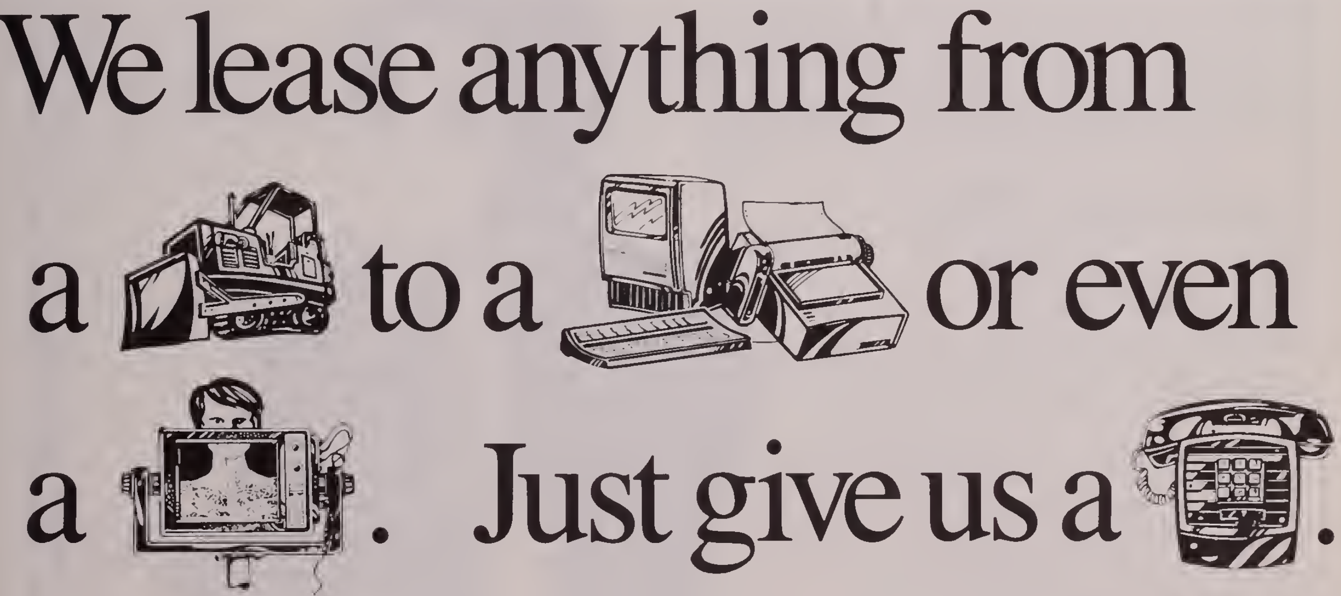

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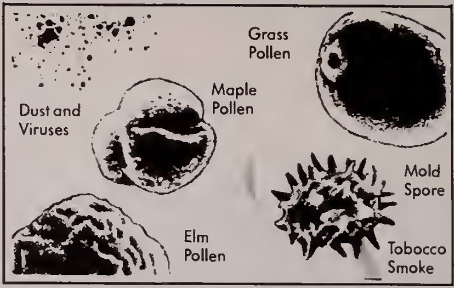

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History of Medicine in Alaska

James S. Pinneo, M.D.

James Pinneo was born into a missionary family which included his mother who served in India and his sister who served as a nurse in Nigeria. While there she contracted Lassa Fever, a highly lethal viral disease. During an emergency flight to the United States, her fever rose as high as 107°F, but she survived. Later she gave plasma to another Lassa fever patient, thus saving his life.

James was born in Newark, New Jersey where his physician father worked in the treatment of poliomyelitis at Children's Hospital. After attending Newark Academy, and spending two years at King's College in Newcastle, Delaware, he was drafted into the Army and was sent to Princeton University for a year of pre-engineering despite requests for pre-medical training. He was with the first division to land at Cherbourg whose harbor he remembers as filled with sunken ships. As an ambulance driver, he drove across Western Europe as far as Czechoslovakia. With death all about, he felt a sense of calm and assurance believing that whatever happened to him was in the Lord's plan.

After the war, he attended Houghton College in pre-med, then went to Temple Medical School still two credits short of a BA degree. He received his MD degree in 1950. His internship at Arlington Memorial Hospital in Pennsylvania was followed by a three-year surgical residency at the Episcopal Hospital in Philadelphia.

In 1951 he married Lois Castor, a nurse. In 1954 they and their two children moved to Glennallen where he joined the independent, non-denominational Central Alaska Mission.

On arrival in Valdez they drove up "the trail," the dirt and mud Richardson Highway which had been constructed during the gold rush. In Glennallen they found a two-room shack where the office also served as a delivery room and mini surgery. Dr. Pinneo relieved the only resident physician, Dr. Chester Schneider for a much needed furlough. When Chet Schneider returned, the two took turns msking monthly trips to Mentasta, Tanacross, Dot Lake, Tok, Northway, Chistochina, and Tetlin. The doctors had staff privileges at Valley Hospital in Palmer where they hospitalized patients.

Vincent J. Joy, the "Flying Missionary," sought to meet the spiritual needs of the people from Valdez to Tok. He was a dedicated man and a skilled pilot.

Keeping cars functional in the Glennallen winter was a major problem and attempts to heat engines with blow torches and camp stoves led to occasional accidents. Once Jim made a house call on an 84-year-old Indian woman living alone in a tent in -65°F weather. She stoked up a fire each morning, chopped wood, and shot a rabbit for breakfast.

Automobile accidents were frequent. One early spring day, Vincent Joy and Jim Pinneo attempted to air evacuate a patient with a compound fracture - dislocation of the hip. The pass south to Valdez was closed, so they attempted to fly north to Fairbanks. At one point they flew 25 feet above a glacier near Delta in a snow squall while Jim administered an IV to the patient in the two-seater plane. They eventually arrived and the patient recovered.

In 1956, Faith Hospital opened as an acute care facility with four beds and a delivery room which was also used for surgery. The four beds were nearly always filled with overflow to the hallways, doctor's offices, or in the case of newborns to bureau drawers. Dr. Pinneo delivered his own third and fourth children. Drs. Schneider and Pinneo saw much tuberculosis, pneumonia, and meningitis. They alternated operating and administering anesthesia, performing appendectomies, herniorrhaphies, and C-sections. Difficult cases were referred to Anchorage or Fairbanks, often with the help of Air Force rescue squadrons.

On one occasion, a man accidentally shot himself in the thigh and abdomen while fishing with a friend in the Tangle Lakes area. They rowed several miles across one lake, portaged to another, and finally walked to a highway roadhouse. Twelve hours after the accident, the patient arrived at Faith Hospital where Jim removed a kidney and ten inches of intestine. The patient recovered.

A walk-in blood bank was set up in the area by Dr. Frank Pauls of the Alaska Department of Health. Dr. Pinneo recalls a woman from Valdez with placenta previa who arrived at the hospital with her husband and three friends, all with blood types matching hers. A live baby was delivered and both mother and baby did well.

The 1964 earthquake devastated Valdez. The greatest damage was done by a tidal wave which came boiling in through the Valdez Narrows bringing a wall of mud and water 85 feet high which destroyed the dock and warehouse and left a freighter on its side. The wave then returned to carry the ship three blocks inland.

One man made his way from the dock area to a trailer park only to see his trailer float out to sea with his wife and daughter. Twenty-eight people in all were swept away. Survivors who flocked to Glennallen were housed in the school and private homes. Faith Hospital had 35 patients, some of whom were in psychological shock. The Army from Fort Wainwright in Fairbanks assisted by providing cots and giving immunizations.

In 1967 Faith Hospital served 8,000 patients. It was enlarged in 1968 by the addition of five more beds and equipment to meet almost any emergency. Money for improvements and for stipends for doctors and nurses came almost entirely from gifts and contributions from church groups. All money received as fees went to overhead. By 1983 the number of outpatients had doubled, but the hospital was closed in 1987 except for an emergency holding facility.

During pipeline construction a school cook came to Faith Hospital with diphtheria. An epidemologic study revealed 15 carriers in Glennallen and eventually nearly three quarters of the people of the state were immunized along with all the inhabitants of two nearby construction camps. One year later a trainee from Glennallen at the Seward Training Center died of diphtheritic myocarditis.

On another occasion, an Alaska Native appeared at the hospital complaining of sore throat and abdominal pain. Shortly after admission he suffered a cardiac arrest, but was resuscitated. After botulism was diagnosed, he admitted to eating "stinkhead soup", made by fermenting fish heads for two weeks, then cooking them into a stew. Dr. Pinneo tested all the other inhabitants of the fish camp and found three more cases. One patient was unconscious for three weeks but eventually all recovered.

Once at Chistochina a 19 year-old was on an antenna tower when a truck backed over a guy wire toppling it. The youth fell 75 feet to the ground and was brought critically injured by heliocopter to Glennallen. He had multiple fractures and no blood pressure. Dr. Pinneo suspected major vessel injuries in the chest. A doctor and a nurse flew him to Anchorage where he received 18 units of blood and was sent on to Stanford where a tear in the root of the aorta was repaired. Three weeks later the patient telephoned Dr. Pinneo to say: "I hear I was in your hospital. Thank you."

Dr. Pinneo recalls an amusing incident in which two persons were seen in a vehicle on the Nabesna Road. After the vehicle returned with only one, investigation revealed "bloody" footprints on the bridge at Slana. After appropriate forensic testing, it was determined that a spruce hen with cranberries in its crop had been shot. The location of the second person was not clarified, but the suspicion of murder ended.

When Dr. Schneider left in 1969 for a psychiatry residency, Jim was left alone at Faith Hospital. Chet Schneider now practices near Philadelphia.

During construction of the TransAlaska pipeline, the mix of patients changed from 1/2 to 1/4 Alaskan Natives. The pipeline workers were slow to pay despite their high wages and often deferred to the union health plans. They were also very demanding. The whole situation was demoralizing to Dr. Pinneo who had conducted his life as a missionary, spreading the message of Jesus Christ and embracing a way of life having little to do with making money.

The pipeline experience which centered on making money was opposite to the philosophy by which James Pinneo lives.

When James Pinneo retired there was a special "Dr. Jim Day" in Glennallen. He was presented with a album picturing the babies he had delivered. Not only had they grown up but also they had brought him a second generation of babies to deliver.

The Pinneos moved to Big Lake in 1987. From there they now travel on short-term missions to Third World countries. They have served in Haiti, Niger and Bangladesh. The picture is always the same: a vast number of preventable diseases including measles, meningitis, pneumonia, malaria, tetanus and infant diarrhea. There are never enough medicines.

Alaska has a history of medical missionaries such as the Moravians at Bethel, Roman Catholics at St. Mary's, Episcopalians on the Yukon, and Presbyterians at Sitka. Missionary commitment and dedication is rarely found now, but James and Lois Pinneo carry on this noble tradition.

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AIR FORCE

THIRD NORTHERN REGIONS CONFERENCE

During September 17 - 21, 1990, Alaska Governor Cowper called the Third International Northern Regions Conference. The meeting was an opportunity for representatives from all over the Arctic and Pacific rims to meet and discuss areas of concern. There was a half day session on Circumpolar Health issues that was chaired by Dr. J.P. Hart Hansen, President of the International Union for Circumpolar Health, the results of which follow:

Summary and Recommendations of the Workshop on Circumpolar Health

There is striking similarity of the health status and health problems that affect residents of all circumpolar countries.

Because of substantial successes during the past four decades in combating certain infectious diseases, life expectancy has improved, infant mortality rates and overall mortality rates have fallen, and overall health status has improved. However, serious problems remain. Health improvements have not occurred equally in all groups, and the health status of arctic indigenous peoples remains much worse than in other populations.

We continue to have problems with mental health, alcohol, tobacco and other substance abuses, suicide, and related social factors such as unemployment and poverty. Also within medical areas like AIDS control, accidents and violence, cardiovascular diseases, maternal and child concerns, and chronic disorders like diabetes and cancer, there are problems.

The main areas of concern include:

1. The special needs of Native peoples;
2. The need for Native peoples to take responsibility of their own lives both individually and collectively;
3. The need for Native peoples to enter the health professions.

Recommendations

We recommend the following for adoption by the Third Northern Regions Conference:

1. That the World Health Organization establish a full time circumpolar desk with a special emphasis on the needs of indigenous populations;
2. That the International Arctic Science Committee establish a special Working Group on Arctic Medical Research represented by the International Union for Circumpolar Health;
3. That the International Union for Circumpolar Health encourage and establish new means of international collaborative research on circumpolar health problems and that further research in basic, environmental and clinical sciences be adequately supported;
4. That the importance of traditional diets and legal access to its sources must be recognized;
5. That adequate and consistent funding for health care delivery in Northern regions and related research be provided in order to eliminate the inequities in health status.

To date, significant activity is taking place toward achievement of recommendation #2. The International Arctic Science Committee (IASC), for which the Secretariat is located in Oslo, Norway, will hold a council/board meeting at the end of January, 1991. At this time the International Union for Circumpolar Health President will present the IUCH proposal for an IASC working group on arctic medical research. Most likely, it will be proposed that this working group could either be the Council of IUCH or rather the Special IUCH Committee on Arctic Medical Research. Dr. Hart Hansen envisions a committee of six to eight members; one or two members of the IUCH council, the rest eminent researchers representing the different circumpolar regions or specific research interests. He proposes the purpose of the Committee should be to handle all relevant questions relating to arctic medical science, to act as scientific advisors to the IASC, and to the organizers of the International Congress on Circumpolar Health, etc.

ALBRECHT-MILAN FOUNDATION OF THE AMERICAN SOCIETY FOR CIRCUMPOLAR HEALTH

The bylaws of the American Society for Circumpolar Health provided for the establishment of an endowment fund entitled the Albrecht-Milan Fund. As is stated in Article II, Section 3:

> "The purposes of the Fund are to raise funds for the Society and to receive and hold by request, grants, and or gifts, any and all funds and expend these funds in the furtherance of the Society's purpose as stated in the Constitution."

The Board of Directors established a Committee to research methods and means of establishing endowment funds. The Board voted to go with a consultant group known for its work in establishing other endowment funds, and directed the Committee to work with the group.

The Albrecht-Milan Endowment Fund committee has met three times since the annual meeting in October, 1990 with special consultant Mr. Kenneth Grounds of the McCormick Group of Williamton, Michigan. The American Society of Circumpolar Health entered into a six month contract with the McCormick Group to assist the committee in developing a foundation and organizing a permanent and on-going giving program.

Progress is being made and the committee with the aid of the consultant is right on the time schedule established at the first meeting.

A name change has taken place and the Albrecht-Milan Endowment Fund has been renamed the Albrecht-Milan Foundation of the American Society for Circumpolar Health (AMF-ASCH). This was done on the advice of the consultant. This broadens the scope for fund raising and enables the AMF-ASCH to attract specific funds for special activities. The broadened concept will demand more of the committee as well as the members of ASCH but it will be worth the effort.

The Articles of Incorporation as well as the Bylaws of the AMF-ASCH have been filed with the State of Alaska.

The original committee (H. Beirne, W. Mills Jr., F. Pauls) has been expanded with the addition of C. Hild, J. Whitlock (CPA) and James Tapscott (Pioneers). The working committee in the next few weeks will be reviewing suggested candidates for the trustees of the AMF-ASCH. There will be trustees representing ASCH as well as trustees with specific skills recruited from the community. All trustees shall receive prior approval from the Board of Directors for ASCH and will serve a three year term.

The organizing committee is also working on the goals and objectives for the AMF-ASCH and developing concept papers. These are examples of research areas where ASCH can exert a leadership role in circumpolar health and are useful in fund raising activities.

At the end of the contract period with the McCormick Group, it is expected that the outcome will be:

1. A fully documented non-profit, tax exempt foundation with all required state and federal (IRS) applications submitted and approved.
2. A fully functioning Board of Trustees, organizationally modeled to meet the identified needs of the ASCH, and with effectively prepared leadership, will be in place.
3. Foundation Trustees will have the "Technology" available to them to carry out the Foundation's fund attraction and management functions.
4. A plan of operations and calendar of activities will be completed and approved by Trustees to attract funds and establish permanent and on-going giving programs.

The formal organization of the AMF-ASCH should be completed by mid-February. At that time members of ASCH will be notified and asked to join in the next step, which is to make the AMF-ASCH a living and dynamic part of ASCH. The committee is doing what was asked by the members and are delivering a strong organization. It is hoped that the members of ASCH will, in turn, participate in the organization's efforts.

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Edited by Robert Fortuine, M.D.

AN EPIDEMIC, A GOVERNOR, AND A SAINT (1819)

In November 1818 a 29-year-old Russian naval officer named Semen Ianovskii was appointed Chief Manager of the Russian-American Company. He had arrived in Sitka in July of the previous year, and seven months later had married Irina, the daughter of the legendary Aleksandr Baranov, who that same month, after nearly 30 years of service, had rather ignominiously been replaced as chief manager.

Ianovskii set out with his wife and infant son Aleksandr in July 1819 for a lengthy inspection tour of the colonies. During his absence an American ship brought a contagious fever to Sitka, whence it spread to Kodiak Island while Ianovskii was visiting St. Paul's Harbor (modern City of Kodiak). Below is his account of the epidemic, which claimed the lives of some 50 persons around the settlement.

Father Herman (or German) was an Orthodox monk who had come to Kodiak with the first group of Russian missionaries in 1794. He later settled on Spruce Island, where he built a school and church, and lived a pious, gentle, and ascetic life in service to the Koniag. He met Ianovskii for the first time on the latter's visit, and their experience together in the epidemic led to a life-long relationship. In 1871, from a seed planted by Father Herman over a half century before, Ianovskii himself became a monk. His mentor was canonized by the Orthodox Church in 1970 as St. Herman of Alaska.

"During my stay on the island of Kadiak [Kodiak] there was brought there a contagious, fatal epidemic disease, or plague. This began with a fever and a heavy cold, a cough, shortness of breath, choking, and three days later death followed! There were neither doctors nor medicines there; the sickness quickly spread throughout the whole settlement and soon crossed to all the nearby islands. The energetic elder Father German ceaselessly, tirelessly and at great personal risk visited the sick, not sparing himself in his role as priest--counselling those suffering to be patient, pray, repent and prepare themselves for death. I, in the course of my duties as administrator and out of sympathy for human suffering, visited them all every day, and took all possible measures to help. I gave advice, gave people tea with elder or camomile, as it came, and issued food--many people were helped by eating warm food--but many were dying. The epidemic affected everyone, even babes in arms; my family: my wife and young babies were also ill, as was I myself. The death rate was so high that after three days there was no one to dig the graves and the corpses lay around unburied. It was good the weather was frosty, because there was no smell.

"I can imagine nothing more somber and terrifying than the spectacle which greeted me when I visited the Aleut *kazhim*! This is a large barn or barracks with board partitions, in which the Aleuts live with their families. In it were almost one hundred people. I visited them all, talking to them, questioning, advising, cheering them up and comforting them. Some were lying already dying, their bodies growing cold, next to those who were still alive; others were already dead: groans and screams tore at my heart. I saw mothers already dead, on whose cold breasts hungry children crawled, crying and trying to find food for themselves, but in vain! My heart contracted with pity, struck with horror, when I saw this melancholy picture of death. No artist could convey in sufficient colors this picture for the illumination of those who are wallowing in luxury, forgetful of death.

"Finally, I myself grew weak and prepared for death; but merciful God had decided to spare me. At night I drank elder-tea, wrapped myself up and sweated, and then I began to feel better. It would seem that only Father German was not affected; for everyone else was, and a good third had died! When the sickness and mortality began visibly to decrease I had already accomplished the business of my visit and I was in haste to return to Sitkha, to the port of Novo-Arkhangelsk, where my permanent residence was and where much important official business awaited me. Now when I bade farewell to Father German, I was doing so to a friend, to my mentor and benefactor. I asked him to pray for me, and set sail."

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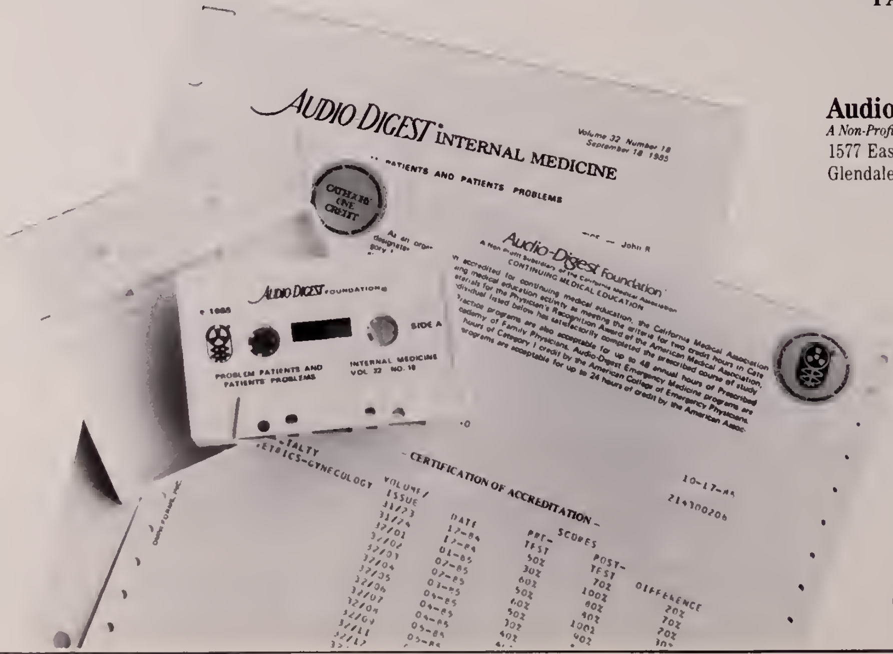

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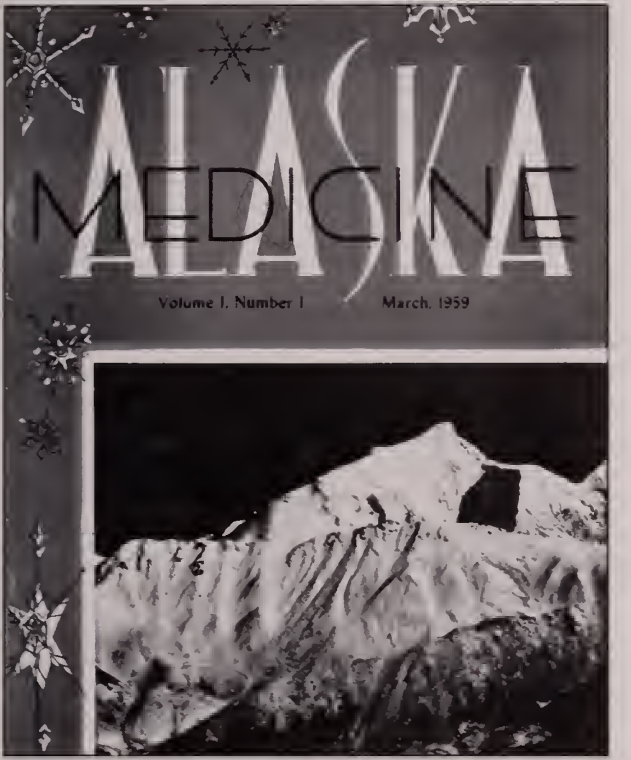

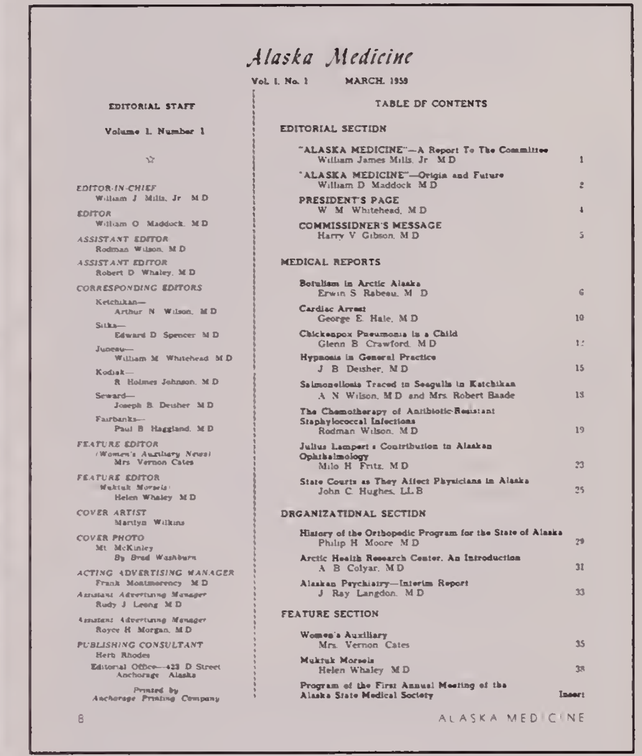

Alaska Medicine

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Volume I, Number 1

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8 ALASKA MEDICINE

The first issue of *Alaska Medicine* was published in March 1959 by the "feasibility committee" appointed May 1958 at the 13th annual meeting of the Alaska Territorial Medical Association. The committee felt the best way to do a feasibility study was to produce the journal to show that it could be done! It was received with delight and was widely acclaimed. Many congratulatory letters were received and only one error pointed out - by Bradford Washborn - that the cover picture was not Mt. McKinley but Mt. Brooks! The first issue was heartily approved by the Alaska State Medical Association March 1959 as the official journal of the association. It was to sell for $1.50 to members and $2.00 for nonmembers. There were to be four issues per year in March, June, September, and December.

Following are excerpts and comments on the first issue of *Alaska Medicine* --- it was truly an outstanding professional journal and truly Alaskan.

* * *

"*Alaska Medicine,* by its demonstration of need, may hasten ideas to further the birth and growth of vitally needed medical programs in our new State. . . . We are given the opportunity now to insure that no longer need the unrecorded medical voice be sent plaintively wailing across the waters of the Inland Passage, to quiver in the Susitna Flats, or be sent rumbling toward the peaks of Mt. McKinley, only to wither and be dissipated in the wilderness. . . . And most important, may our journal provide us with such content, that from its pages we can continue the process of learning, and develop further the process of thinking. We hope by its quality and purpose, that someday, physicians elsewhere, as have navigators for centuries, will look upward, and to the North, to Polaris and to the 49th Star, and in those latitudes find light." *William J. Mills, Jr., MD for the committee, Alaska Medicine, March 1959.*

"At first thought it might seem overly amibitious for Alaska with scarcely 100 practicing physicians to have a state medical journal. Many states with physicians numbering in the thousands do not have a medical publication. Cogent reasons exist, however, for us to have our own journal. There is a pressing need for communication among the handful of physicians in Alaska, the largest state. The extremes of arctic and sub-arctic weather present special medical problems not encountered in the "South 48." The responsibilities of newly acquired statehood present organizational issues directly affecting physicians and their patients, issues which would be worthy of discussion in our own medical publication." *William O. Maddock, MD, editor, Volume 1, Number 1, Alaska Medicine.*

"...during my second term as your President, we are considering the possibility of having our own official State Journal. . . we have come a long way in a period of a few years. When I was elected President in 1944, I elected myself. There were no meetings and the elections were held by mail.... I cast the tie-breaking vote for myself. In 1946 we had our first organized meeting here in Juneau with Dr. A.N. Wilson, Ketchikan, as our President. A lot of credit for this organized meeting must be given to Dr. C. Earl Albrecht, who was then Commissioner of Health." *W.M. Whitehead, M.D. President ASMA 1958-1959.*

"Congratulations and success from the Alaska Dept. of Health to *Alaska Medicine*. Its birth marks a great forward step in Alaska medical affairs and its maturity will enable a development and synthesis of medical opinions among Alaskan members of medical disciplines which is a rarity under such circumstances." *Harry V. Gibson, M.D. Commissioner of Health - State of Alaska.*

* * *

There were very timely excellent medical reports on Botulism, Cardiac Arrest, Chickenpox, Pneumonia, and Salmonellosis from seagulls. To a new physician in Alaska, this was fascinating to me as I had not seen such cases in medical school and common childhood diseases were simply common and not fulminant as was often the case in 1958 Alaska. No physician in Alaska will forget the first case of botulism seen and Dr. Hale's article on cardiac arrest (". . . a bold opening of the chest . . .") certainly brought back memories on the development of CPR -- reminiscent of the days when some medical schools ordered students and house staff to carry a sharp knife at all times. (I still have mine!)

Space does not allow me to comment on all the medical reports nor on the very interesting organizational section; e.g., the Orthopedic Program developed by Dr. Philip Moore. (His first statewide survey in 1946 revealed 440 critical cases needing hospitalization --initially most were due to Tuberculosis.) Dr. A.B. Colyar presented a historical review of the development and role of the Arctic Health Research Center started in 1948 regarding zoonotic diseases, tuberculosis, toxic clams, fish tapeworm, and congenital methemoglobinemia. (Again, never forgotten is the first case of a seemingly healthy but cyanotic patient.) Dr. J. Ray Langdon reviewed the status of the mental health program for Alaska. I think there were only two or three psychiatrists in the whole state at that time and patients needing hospitalization were sent "Outside."

* * *

"Dr. and Mrs. Vincent Hume and family of Palmer are in good health following an experience they will never forget. While driving across a local lake, the ice broke and their car plunged into the water. Fortunately the family of seven including a small baby reached safety before the car went to the bottom". *An item from the Women's Auxiliary report by Mrs. Vernon Cates.*

Muktuk Morsels by Helen Whaley, M.D. excerpts: "Statehood has caused a tremendous surge of interest in the practice of medicine in Alaska. Articles have appeared in such diversified publications as the *New England Journal of Medicine*, which presented an editorial on Alaska's health for the past 10 years based on Metropolitan Life Insurance statistics; *Medical Economics* which gave a fairly complete survey of the coverage and opportunities for practice in Alaska, *Scope* newspaper which had a pictorial review of Dr. Milo Fritz and Dr. Rambo of New York City surveying the ENT problems among the Alaska Natives and more recently, *Time* magazine, which gave a detailed report of the method of diagnosis and treatment via radio used by Dr. William Brownlee and Dr. Jean Persons of the USPHS at Bethel. There have been several post-graduate sessions including the Lederle Laboratory Seminar in Anchorage 2/21/1959. (Was this the 1st or 2nd annual Lederle Symposium?) In September 1958, through the joint cooperation of the Alaska Heart Association and the Alaska Department of Health, cardiac clinics were held in Anchorage and at Mt. Edgecumbe, Sitka, with Drs. Gerbode, Hultgren and Hochstetler of Stanford University. (Was this the 1st annual cardiac clinic?) Approximately 100 patients of all ages with congenital and acquired heart disease were evaluated. The clinics were held at the USPHS hospitals. Because of the increase in poison ingestion both in the larger communities and in the more remote bush areas, a need for a central poison control center became obvious. Currently a center is being established at the Northward Drug Store in Fairbanks by Mark Fenton, Ann Berrigan and Mell Allen; and in the outpatient department at the Anchorage USPHS hospital by Dr. Gloria Park. (I started out with 2 books and about 500 unorganized file cards from the Washington, D.C. Poison Control Information Center.)

* * *

Fairbanks - Dr. & Mrs. James Lundquist had a baby girl October 1958. Dr. Paul Hagglund became a fellow of the American College of Surgeons. Dr. Andrew Schaible married Grace Bergh and they flew over the pole to visit South Africa.

Bethel - Dr. Jean Persons had a baby girl January 1959. Bethel will have its first private physician when Dr. Harriet Jackson (Shirmer) returns this summer.

Dillingham - Dr. George Wagnon (USPHS) recently flew his Stinson station wagon to Anchorage for repairs. Dr. John Libby of Goldendale, Washington plans to enter private practice in Dillingham.

Glenallen - Dr. James Pinneo has returned to Faith Hospital.

Kenai - Dr. Marion Goble is the first private physician to settle in the Kenai-Soldotna area.

Anchorage - Following post-graduate work, Dr. Howard Romig has returned to the practice of OB-GYN. Dr. Merritt Starr has been on a world tour. Dr. Nancy Sydnam had a baby boy. Dr. Lester Margetts is Chief of Staff at Providence. Dr. William J. Mills, Jr. is President of the Anchorage Medical Society. Dr. William Rader plans to leave for a psychiatry residency.

Juneau - Dr. Henry Wilde, who attended high school in Juneau, has returned as an internist.

Sitka - Dr. Tillman Moore, Jr. has joined Dr. Philip Moore (no relation).

Ketchikan - Dr. A.N. Wilson's son, Jim, is a surgical resident and son, Arthur Jr. is an intern.

* * *

Last but not least I'd like to comment on the numerous ads in the first issue of *Alaska Medicine.* These ads are of considerable historical note with now obsolete items and businesses, old dress styles and even an ashtray on a doctor's desk. There were several ads not directly medically related eg. banks, insurance company, travel agency, laundries and Pacific Northern Airlines with three Lockheed Constellation flights to Seattle.

Without the support of numerous pharmaceutical and business ads, I doubt the first issue of Alaska Medicine would have been published. Much thanks is due them for their support over the years.

If you're interested in history, come into the Alaska State Medical Association office and browse through the old journals. If you have old copies in the attic you can spare, bring them along as the office collection is incomplete. I have thoroughly enjoyed reviewing and writing about Volume 1, Number 1 of *Alaska Medicine* and I plan to continue this column if there is enough interest in "Out of the Past . . .".

RURAL HEALTH:

Harvesting Our Experience

National Rural Health Association
14th Annual National Conference
May 19–22, 1991
Seattle, Washington

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Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

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1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
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4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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RESOLUTIONS

ADOPTED AT THE ALASKA STATE MEDICAL ASSOCIATION COUNCIL MEETING
Anchorage, Alaska

October 20, 1990

Resolution : 90-21
Subject: Illegal Breath Alcohol Concentrations for Driving

Whereas, death and injury continue on the nation's highways because of alcohol-impaired drivers; and

Whereas, an important law enforcement procedure involves measurement of alcohol in expired air even though most states set limits based on blood alcohol concentrations; and

Whereas, the variable relationship between blood and breath alcohol concentrations often works to the advantage of the drunk driver's defense; therefore be it

RESOLVED, That the American Medical Association urge states to amend their appropriate laws to deem driving illegal with a breath alcohol concentration greater than .05 gms of alcohol per 210 L of expired air or .05 gms alcohol per 100 cc whole blood.

No fiscal impact.

Resolution 90-22
Subject: Driving Under the Influence

Whereas, death and injury happens when driving while intoxicated; and

Whereas, many states entitle the offense of drunk driving as "Driving While Intoxicated" (DWI); and

Whereas, the definition of "intoxicated" is imprecise and argument over this definition often works to the advantage of the drunk driver's defense; therefore be it

RESOLVED, That the American Medical Association urge states to retitle the crime "Driving Under the Influence" (DUI) since one is under the influence of however much one has consumed, regardless of outward signs of "intoxication".

No fiscal impact.

Resolution 90-23
Subject: Gun Control

Whereas, gun control is a highly controversial and emotionally laden issue; and

Whereas, many physicians, at least in Alaska, are hunters, gun collectors and competitive shooters; and

Whereas, some political issues are outside the area of expertise of physicians and should therefore be "off limits" to activism; therefore be it

RESOLVED, That the American Medical Association policy be revised to reflect neutrality on gun control as a concept, while still deploring improper use of guns.

No fiscal impact.

Resolution: 90-24
Subject: Memorial Resolution for Joseph M. Ribar, M.D.

Whereas, family, friends and colleagues were deeply sorrowed by the loss of Joseph M. Ribar, M.D. on October 12, 1990; and

Whereas, Doctor Ribar cultivated broad interests and served in a variety of professional roles as physician, teacher, civic leader, spokesman, organizer and administrator; and

Whereas, he practiced the specialty of Family Practice medicine in the community of Fairbanks, Alaska, for 37 years, acknowledged and respected by his peers; and

Whereas, he held the office of Mayor of Fairbanks, Alaska, the office of Borough Council Member, and was an active community leader; and

Whereas, he was a member of the Alaska State

Medical Association, council member of the Alaska State Medical Association, Alaska delegate to the American Medical Association for 20 years, member of the American Academy of Family Practice, and vice president of the American Medical Association; and

Whereas, he was appointed by President Lyndon B. Johnson to the National Council on Aging, and in 1986 was one of 10 doctors nominated for the American Academy of Family Physicians' "Family Doctor of the Year" award; and

Whereas, Doctor Ribar lived with distinction, dedication and devotion; therefore be it

RESOLVED, That the heartfelt sympathy of the House of Delegates be conveyed to his family.

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In Memorium

MOHAMMAD SARWAR, M.D,

On July 16, Alaska medicine lost one of its finest with the passing of Mohammed Sarwar. His death came just ten weeks after he had carried out an unprecedented series of cardiac operations, despite the onset of terminal pain that characterized his final course. Nevertheless, his results were of the excellent quality that the medical community had come to expect from his record of achievement.

Born of humble beginnings, in Sharwal, Pakistan in 1948, Mohammed Sarwar, ("Mo", as he was affectionately known to most) left his parents at the age of 10 to join his older brother, Mohammed Anwar, in Manchester, England. There, with his brother's fostering and support, he pursued his education and initial medical training. He entered Manchester University in 1967, after completing requirements at the Lancashire and Cheshire Institutes, achieving distinction in Chemistry, Physics, and "Engineering Workshop". Medical school was completed at the Victoria University of Manchester, with the earning of his MBChB degree in 1972. Mo served as a house officer and taught anatomy at his alma mater for two years before leaving England for Ohio, where he again interned in 1974-75. From 1975 to 1982, he pursued residency training in General and Thoracic Surgery in Houston at the Baylor College program directed by Dr. Michael E. DeBakey. When he set out for Anchorage, in 1982, Mo had licenses to practice in Ohio, Washington D.C., Texas, and Alaska. He was certified by the American Board of Thoracic Surgery in 1983, and he was made a Fellow of the American College of Surgeons in 1986. Despite his youth, the esteem with which he was regarded by his colleagues resulted in his chairing the Department of Surgery at Providence Hospital in 1988.

Shortly after his arrival in Anchorage, and for most of his last six years, Dr. Sarwar served as the only cardiac surgeon in Alaska. His accomplishment of virtually uninterrupted service in this capacity was exceeded only by the consistency of his superior results. His commitment saw him seldom out of the hospital, and rarely away form Anchorage. This tragically took a toll in his personal life, and limited his opportunity to capitalize on many of his talents.

I first met Mo at a small meeting where his quiet manner and restraint were immediately registered when his economy of words and thoughtful pertinence commanded attention. Later, observing him at surgery became an opportunity to witness the exemplary abilities of a master "beyond his years". He was patient, thoughtful, deliberate, efficient, and exacting. He practiced with a respect for basic-science principles in the operating room and Hippocratic principles in his decision making and involvement with patients and colleagues alike. He seemed to possess an uncanny ability to predict from among those patients being considered for surgery, those who would fair well with medical rather than surgical treatment, and he would not be hasty in moving patients along to elective operations. His skills in the operating room reflected the best of his surgical alma mater. His manner, and his deportment and demeanor represented a disregard for the "glitz" of the same institution.

A steady and methodical pace was his characteristic. His memory was enviable. Mo rarely kept a patient list and remembered patients and their problems for years.

Mo loved the arts and particularly classical music. He enjoyed solving mechanical problems and when given the luxury of time, he became engrossed in the mastery of any new and unfamiliar subjects. Return visits to England drew him, as did his special retreat near Talkeetna. Unfortunately, he was seldom able to indulge in interests outside of his professional activities.

Mo was of very temperate disposition. He did, however, prefer highly seasoned food, used cigarettes for relief, and substituted coffee for nutrition. Despite this, he had the steadiest hands I have ever seen in an operating room. One might, however, speculate that his premature demise was in part at least, attributable to these few weaknesses.

Mo was revered by all who worked with him in the operating room, intensive care, and the wards. His even temperament and unique sense of humor endeared him and he was generous in expression of appreciation to those on whose help he relied. His pizza parties after long days operating became a tradition. Ironically, acute symptoms during his last such event led to the diagnosis of his ultimately fatal illness.

(Continued on page 166)

JOSEPH M. RIBAR, M.D.

1923-1990

"Joe" to his colleagues and to the AMA, "Dr. Joe" to his patients, "DR JMR" to the Department of Motor Vehicles, left us 12 October 1990, after a long illness detected and determined to be fatal during the Alaska State Medical Association meeting in June.

Though Joe was 'competition' these many years since his arrival in Fairbanks 35+ years ago, he and I were the best of friends in large part because we thought alike in the practice of medicine and in medical politics. For me, and I am certain for all of his colleagues in medicine, Joe's death is a bitter loss.

I prefer to avoid funerals, but did go to Joe's; and I barely survived. In general, I've felt like Charles Lamb, "Anything terrible makes me laugh; I once misbehaved at a funeral," but Joe's funeral did not bring me to laughter; rather I was in tears throughout.

Joe was one of the last of the true general practioners, taking care of the whole patient; he objected to the dilution of the AAGP to the AAFP,staffed with physicians not wholly trained or experienced. He believed that the best specialists were those who had a general practice background; they were whole physicians, "real doctors". Without asking for it he was held in reverence by his patients, because he was a friend and a competent, caring GP.

He served his community as mayor and in other capacities; he achieved a balance of work and play and community service. Much of his time over all these years was spent supporting and representing, in the AMA, the physicians of Alaska. All of Alaska's physicians, not only those who were members of the Alaska State Medical Association, owe Joe a tremendous debt.

In his delegate responsibilities with the AMA Joe worked effectively and extremely hard; he was known and respected by all in the house of delegates and in the administration of the AMA. One of the very effective ways to lose weight was to go to an AMA business session and try to keep up with Joe. He'd run from 6:30 A.M. one day until 1:00 or 2:00 A.M. the next. Joe did this year after year, and he left his imprint upon the national organization as well as upon the state and local medical organizations.

We're all indebted to Joe, and all of us will miss his efforts on our behalf.

Thanks, Joe, and good-bye.

James A. Lundquist, M.D.

BILL IVY - MY FRIEND

On August 18, 1990, Anchorage lost a great man and a great physician - Bill Ivy. Bill practiced in Anchorage as an OB/GYN and Family Practice physician for over 40 years. He was instrumental in building up the medical establishment in Anchorage.

I first met him 26 years ago when I joined him and 6 other physicians in the Doctors Clinic. Several years prior to that time, he and several of his clinic partners had mortgaged their homes to buy land to build a new hospital. This land was across the street from the old Providence Hospital on 8th and L Street and is currently the Municipal Health Building. The dream in those days was to build a "Mayo Clinic of the North" to provide first rate care for the people of Anchorage. Over the 40 years, the clinic grew and grew and then it failed. The hospital grew and grew, changed names several times, and succeeded. A new hospital was then built on DeBarr Road which is currently known as the Humana Hospital-Alaska.

Over these years of upheaval and change, Bill was a constant and steady force in promoting the upgrading of medical care in Anchorage. He served as Chief of Staff of the hospital as well as president of the clinic for many years. He championed bringing new specialists and new technology into the city. He meanwhile maintained his expertise throughout and prided himself on keeping abreast of the newest procedures and treatment. The administrative work involved his having to be present at many interminable meetings. He had a unique way of sorting out priorities and influencing his peers to follow him. He would stand up to speak, wait for all the noise to die away, and then he would speak very softly and slowly. Invariably, however, he came right to the heart of the problem. His opinions were most often right on target and were respected by his peers. On the few occasions where his powers of persuasion were insufficient to save the day, he could spring into action and do things with a pragmatic hands-on approach.

He was a builder, not only of hospitals and clinics, but also of homes and cabins. While all this was going on, he maintained an active practice-delivering hundreds of babies and had a legion of faithful patients who considered him their friend as well as their physician. He was a pilot, fisherman, hunter. He was truly the Gentle Giant of Anchorage physicians. In later years,

(Continued on page 166)

President's Corner

Alas! The Magadan meeting is not to materialize. The recent Minister of Health, Dr. Lebedev, was transferred to Moscow, presumably a promotion, but you never know. His successor has not been to my house for dinner yet, but the new deputy minister, Dr. Valeri Sauhat, has. He tells us that the economy of Magadan is a basket case, that the hospital construction project is on hold, that the new equipment they purchased is still in the crates, and that food is scarce and lodging is nonexistent. He hopes that in two or three years conditions will improve to the point where we can again consider a joint meeting there, but again, long range plans are difficult for them to make.

As an alternative, Dr. Sauhat suggests that we might have a joint meeting in Khararovsk. It would really be a tourist trip with enough medicine thrown in to satisfy the IRS. We are investigating this possibility - maybe we can put something together for 1992 or so. More anon.

Meanwhile, keep practicing your Russian.

Donald R. Rogers, M.D.
President
Alaska State Medical Association

(Continued from page 164)

He remained strong of disposition through his last eight months, inspiring all with his courage and selfless concern for the hospital program that had long been his singular charge.

Mohammed Sarwar passed away quietly with friends and closest relatives nearby. His body rests in Manchester, the distant home of his relatives, and the source of his western education and culture. His spirit will live long in Alaska with those of us who loved him for his great skill, many qualities and his commitment to Alaska and the care of her people.

R. Leighton Fisk, M.D.

(Continued from page 165)

he turned to art with the same intensity that he had exhibited in medicine. Of his many accomplishments, I feel one of the most important was the genesis and nurturing of an alternative first rate hospital in Anchorage so that patients and physicians alike had a choice.

Thanks again, Bill, for all you've done for Alaska.

Your friend,

Sam DePalatis, M.D.

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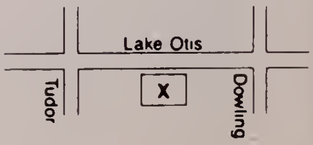

Letters to the Editor

ELIMINATION OF PREVENTABLE BLINDNESS FROM DIABETES BY THE YEAR 2000

Dear Editor:

In no field of medicine is continuing medical education of physicians more critical than in the management of diabetes. Twelve million Americans have diabetes mellitus, and the disease knows no medical specialty boundaries. In addition to being a major cause of morbidity from multi-system complications, such as renal failure, neuropathy, and cardiovascular disease, diabetes is the leading cause of blindness among working-age Americans. It accounts for at least 12 percent of new cases of blindness every year in the United States. Diabetic retinopathy is often asymptomatic at its most treatable stage, emphasizing the importance of early diagnosis of this retinal complication. New information about diabetes emerges almost monthly as published in some specialty journal, making it mandatory for us to find better ways to "keep up" with advances in this field -- the stakes are simply too high to do otherwise.

In response to the increasing importance of the overall problem of diabetes and diabetic retinopathy, and the availability of improved treatment regimens defined by published clinical trials result, the American Academy of Ophthalmology (AAO) has embarked on a long-term education project designed to more rapidly translate research findings to medical benefits for the American public. The new project -- "Elimination of Preventable Blindness from Diabetes by the Year 2000" -- or "Diabetes 2000" -- was announced at the AAO's 1989 Annual Meeting.

FACTS ABOUT DIABETIC RETINOPATHY

The majority of diabetic patients have non-insulin-dependent diabetes mellitus (NIDDM, Type II). Usually diagnosed after age 40, Type II diabetic patients may or may not be treated with insulin. Fewer patients have insulin-dependent diabetes mellitus (IDM, Type I), which is usually diagnosed before age 30. type I diabetic experience more frequent and severe ocular complications. After five years, 23 percent of Type I diabetic patients have retinopathy; after ten years, almost 60 percent have retinopathy; and after 15 years, 80 percent have it. Proliferative diabetic retinopathy (PDR) - the most threatening form of retinopathy - is present in 25 percent of Type I patients after 15 years and often remains asymptomatic well beyond the optimal stage for treatment.

An estimated 700,000 Americans have proliferative diabetic retinopathy and 500,000 have macular edema. The annual projected incidence of new cases of PDR and macular edema is 65,000 and 75,000, respectively. About 8,000 new cases of blindness a year in the United States are caused by complications from diabetes.

WHAT CAN BE DONE

"Diabetes 2000" provides the means to close the gap between advances in research and changes in treatment patterns. While most physicians are aware of diabetic retinopathy, the AAO's goal now is to focus attention on the importance of early diagnosis and timely treatment of diabetic retinopathy based on the important advances of the last 5-10 years. For example, we now know that timely laser photocoagulation surgery can reduce the risk of visual loss from proliferative diabetic retinopathy by at least 50 percent. We know that timely laser photocoagulation surgery of diabetic macular edema can reduce the risk of moderate visual loss by 50%. We know that vitrectomy surgery can restore useful vision to some diabetic patients who have advanced diabetic retinopathy.

EFFECTIVE PARTNERSHIPS ARE NEEDED

As the working title implies, "Diabetes 2000" will be a long-term project aimed at a specific disease: diabetic retinopathy and its complications. Several phases are anticipated. Providing the latest research findings to ophthalmologists and other physicians who care for those patients is the first priority, followed by patient and public information. Initially, new advances and treatment guidelines for the medical and surgical treatment of diabetic eye disease will be emphasized through continuing education of ophthalmologists, other physicians, allied health professionals, residents, medical students, and specialists in diabetes-related education. In later phases, education programs for diabetic patients and the public will be developed.

Ultimately, improved eye care of diabetic patients is expected as a result of closer collaboration between the physician primarily responsible for the care of the patient's systemic illness, the patient, and the ophthalmologist. Since diabetic patients can be asymptomatic despite significant progression of diabetic retinopathy, the importance of a renewed and improved partnership between the ophthalmologist and the patient's primary physician is critical.

Because of the ambitious goal and long time-frame, many other medical organizations and public groups are involved in Diabetes 2000. Representatives from various medical specialties, government agencies, and other organizations devoted to problems of the diabetic patient have been invited to participate. Since diabetes is a complex, multi-system disease, whose overall management is the responsibility of physicians other than ophthalmologists, "Diabetes 2000" will stress involvement of other physicians and medical specialty organizations in the planning and implementation of the project. The importance of finding ways to develop effective partnerships between the patient's primary physicians, the ophthalmologist, and the patient in the management of diabetic eye disease is a major goal. Another important aspect of the project is the identification and promotion of existing diabetic eye health programs around the country, such as the Center for Disease Control (CDC) Diabetes Translation Project. The AAO will encourage ophthalmologists to participate in national, regional, and local programs that are already in operation, as sponsored by such organizations as the American Diabetes Association, the Juvenile Diabetes Foundation, Lions Clubs, and others.

ALASKA OPHTHALMOLOGISTS ARE PARTICIPATING

Education materials are being developed and demonstration projects are underway in some states to encourage ophthalmologists and other physicians to participate in continuing education programs concerned with the overall management of diabetic retinopathy. A Preferred Practice Pattern on diabetic retinopathy is available through the offices of the American Academy of Ophthalmology (415-561-8500). this document provides the latest information concerning the management of diabetic retinopathy. The Alaska State Ophthalmological Society is actively involved in this national initiative.

Ophthalmologist in Alaska are eager to help develop the necessary education and service programs that will achieve this goal. Diabetes 2000 will parallel a major diabetic retinopathy public information campaign recently announced by the National Eye Institute. The NEI's National Eye Health Education Program (NEHEP), which targets both diabetic retinopathy and glaucoma, is fashioned along the lines of earlier federal initiatives against smoking and high-blood pressure. By continuously updating our medical knowledge and skills related to this multi-system disorder, and by forging partnerships between physicians in the effective and efficient management of diabetic patients, we have a unique and important opportunity - we can rescue preventable blindness from diabetes by the year 2000.

Marvin Grendahl, M.D.
President
Alaska State Ophthalmoligical Society

Ronald E. Smith, M.D.
AAO Secretary for Continuing Education
Diabetes 2000 National Project Director

Arnall Patz, M.D.
Past President, AAO
Diabetes 2000 National Chairperson

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"KEEPING YOUR DOCTOR IN STITCHES"

Dear Editor,

After twenty-three years of reporting the serious side of medicine, for Westinghouse Broadcasting in Los Angeles, I am currently writing a book on the lighter side. The book, tentatively titled "Keeping Your Doctor in Stitches" deals with true incidents that take place in a physician's practice, at his or her office or in the hospital.

Initial reaction to this book has been extremely positive from physicians all across the country, and I would like to be able to include stories from your state.

You could be of great assistance to me in this project if you would include a short item in your publication, about the stories I am after.

If you would like more information, check the April 20th 1990 issue of American Medical News, page 27, for a full story.

Don Herbert
12327 Erwin Street
North Hollywood, California 91606

TOPIC: THEOPHYLLINE TOXICITY

Dear Editor,

I have recently become aware of a significant problem with theophylline about which physicians may not be informed; the alteration of theophylline clearance during viral illness or fever. Serum levels can be increased from the therapeutic range into the toxic range by either viral illness or fever. The majority of physicians in my community were not aware of the seriousness of this problem. I hope to share this information as well as the current philosophy regarding theophylline.

Please help by forwarding the enclosed memorandum on theophylline toxicity to your physician members.

James A. Klicpera, M.D.
The Everett Clinic
3901 Hoyt Ave.
Everett, WA 98201

Theophylline toxicity resulting from overdose has been recognized for a long time as a potential problem when using this drug in the management of reactive airways disease. It is now recognized that elevated temperature lasting 24 hours or longer and probably many viral infections can significantly alter the clearance of theophylline resulting in elevation of serum theophylline levels into the toxic range. Seizures with permanent severe brain damage may occur as a result of high serum theophylline levels. Please have have your members who use this drug review the medical literature regarding the safe use of theophylline. Consider using other medications first before using theophylline. If theophylline is used, keep serum levels between 5-15 ug/ml instead of the 10-20 ug/ml range and should fever or viral infections of 24 hours duration or longer occur reduce the dose of theophylline by ½ during that illness or if that is not safe then monitor theophylline levels more carefully.

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VASOTEC®

(ENALAPRIL MALEATE | MSD)

VASOTEC is available in 2.5-mg, 5-mg, 10-mg, and 20-mg tablet strengths.

Contraindications: VASOTEC® (Enalapril Maleate, MSD) is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor.

Warnings: *Angioedema* Angioedema of the face, extremities, lips, tongue, glottis, and/or larynx has been reported in patients treated with ACE inhibitors, including VASOTEC. In such cases, VASOTEC should be promptly discontinued and the patient carefully observed until the swelling disappears. In instances where swelling has been confined to the face and lips, the condition has generally resolved without treatment, although antihistamines have been useful in relieving symptoms. Angioedema associated with laryngeal edema may be fatal. **Where there is involvement of the tongue, glottis, or larynx likely to cause airway obstruction, appropriate therapy, e.g., subcutaneous epinephrine solution 1:1000 (0.3 mL to 0.5 mL), should be promptly administered.** (See ADVERSE REACTIONS.)

Hypotension: Excessive hypotension is rare in uncomplicated hypertensive patients treated with VASOTEC alone Patients with heart failure given VASOTEC commonly have some reduction in blood pressure, especially with the first dose, but discontinuation of therapy for continuing symptomatic hypotension usually is not necessary when dosing instructions are followed, caution should be observed when initiating therapy. (See DOSAGE AND ADMINISTRATION.) Patients at risk for excessive hypotension, sometimes associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death, include those with the following conditions or characteristics: heart failure, hyponatremia, high-dose diuretic therapy, recent intensive diuresis or increase in diuretic dose, renal dialysis, or severe volume and/or salt depletion of any etiology It may be advisable to eliminate the diuretic (except in patients with heart failure), reduce the diuretic dose, or increase salt intake cautiously before initiating therapy with VASOTEC in patients at risk for excessive hypotension who are able to tolerate such adjustments. (See PRECAUTIONS, *Drug Interactions* and ADVERSE REACTIONS.) In patients at risk for excessive hypotension, therapy should be started under very close medical supervision and such patients should be followed closely for the first two weeks of treatment and whenever the dose of enalapril and/or diuretic is increased. Similar considerations may apply to patients with ischemic heart disease or cardiovascular disease in whom an excessive fall in blood pressure could result in a myocardial infarction or cerebrovascular accident. If excessive hypotension occurs, the patient should be placed in the supine position and, if necessary, receive an intravenous infusion of normal saline. A transient hypotensive response is not a contraindication to further doses of VASOTEC, which usually can be given without difficulty once the blood pressure has stabilized. If symptomatic hypotension develops, a dose reduction or discontinuation of VASOTEC or concomitant diuretic may be necessary

Neutropenia/Agranulocytosis: Another ACE inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression, rarely in uncomplicated patients but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease. Available data from clinical trials of enalapril are insufficient to show that enalapril does not cause agranulocytosis at similar rates. Foreign marketing experience has revealed several cases of neutropenia or agranulocytosis in which a causal relationship to enalapril cannot be excluded Periodic monitoring of white blood cell counts in patients with collagen vascular disease and renal disease should be considered

Precautions: *General: Impaired Renal Function:* As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including VASOTEC, may be associated with oliguria and/or progressive azotemia and rarely with acute renal failure and/or death.

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine were observed in 20% of patients. These increases were almost always reversible upon discontinuation of enalapril and/or diuretic therapy In such patients, renal function should be monitored during the first few weeks of therapy

Some patients with hypertension or heart failure with no apparent preexisting renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when VASOTEC has been given concomitantly with a diuretic. This is more likely to occur in patients with preexisting renal impairment. Dosage reduction and/or discontinuation of the diuretic and/or VASOTEC may be required.

Evaluation of patients with hypertension or heart failure should always include assessment of renal function. (See DOSAGE AND ADMINISTRATION.)

Hyperkalemia: Elevated serum potassium (>5.7 mEq/L) was observed in approximately 1% of hypertensive patients in clinical trials. In most cases these were isolated values which resolved despite continued therapy. Hyperkalemia was a cause of discontinuation of therapy in 0.28% of hypertensive patients. In clinical trials in heart failure, hyperkalemia was observed in 3.8% of patients, but was not a cause for discontinuation.

Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with VASOTEC (See *Drug Interactions.*)

Surgery/Anesthesia. In patients undergoing major surgery or during anesthesia with agents that produce hypotension, enalapril may block angiotensin II formation secondary to compensatory renin release If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion.

Information for Patients.

Angioedema Angioedema, including laryngeal edema, may occur especially following the first dose of enalapril. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to take no more drug until they have consulted with the prescribing physician.

Hypotension. Patients should be cautioned to report lightheadedness, especially during the first few days of therapy If actual syncope occurs, the patients should be told to discontinue the drug until they have consulted with the prescribing physician.

All patients should be cautioned that excessive perspiration and dehydration may lead to an excessive fall in blood pressure because of reduction in fluid volume. Other causes of volume depletion such as vomiting or diarrhea may also lead to a fall in blood pressure; patients should be advised to consult with the physician.

Hyperkalemia: Patients should be told not to use salt substitutes containing potassium without consulting their physician.

Neutropenia: Patients should be told to report promptly any indication of infection (e g., sore throat, fever) which may be a sign of neutropenia.

NOTE As with many other drugs, certain advice to patients being treated with enalapril is warranted This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects.

Drug Interactions:

Hypotension: Patients on Diuretic Therapy: Patients on diuretics and especially those in whom diuretic therapy was recently instituted may occasionally experience an excessive reduction of blood pressure after initiation of therapy with enalapril. The possibility of hypotensive effects with enalapril can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with enalapril. If it is necessary to continue the diuretic, provide close medical supervision after the initial dose for at least two hours and until blood pressure has stabilized for at least an additional hour (See WARNINGS and DOSAGE AND ADMINISTRATION.)

Agents Causing Renin Release: The antihypertensive effect of VASOTEC is augmented by antihypertensive agents that cause renin release (e g , diuretics).

Other Cardiovascular Agents: VASOTEC has been used concomitantly with beta-adrenergic-blocking agents, methyldopa, nitrates, calcium-blocking agents, hydralazine, prazosin, and digoxin without evidence of clinically significant adverse interactions.

Agents Increasing Serum Potassium: VASOTEC attenuates potassium loss caused by thiazide-type diuretics. Potassium-sparing diuretics (e.g , spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium. Therefore, if concomitant use of these agents is indicated because of demonstrated hypokalemia, they should be used with caution and with frequent monitoring of serum potassium. Potassium-sparing agents should generally not be used in patients with heart failure receiving VASOTEC.

Lithium: Lithium toxicity has been reported in patients receiving lithium concomitantly with drugs which cause elimination of sodium, including ACE inhibitors A few cases of lithium toxicity have been reported in patients receiving concomitant VASOTEC and lithium and were reversible upon discontinuation of both drugs. It is recommended that serum lithium levels be monitored frequently if enalapril is administered concomitantly with lithium.

Pregnancy – Category C: There was no fetotoxicity or teratogenicity in rats treated with up to 200 mg/kg/day of enalapril (333 times the maximum human dose). Fetotoxicity, expressed as a decrease in average fetal weight, occurred in rats given 1200 mg/kg/day of enalapril but did not occur when these animals were supplemented with saline. Enalapril was not teratogenic in rabbits. However, maternal and fetal toxicity occurred in some rabbits at doses of 1 mg/kg/day or more. Saline supplementation prevented the maternal and fetal toxicity seen at doses of 3 and 10 mg/kg/day, but not at 30 mg/kg/day (50 times the maximum human dose).

Radioactivity was found to cross the placenta following administration of labeled enalapril to pregnant hamsters.

There are no adequate and well-controlled studies of enalapril in pregnant women. However, data are available that show enalapril crosses the human placenta Because the risk of fetal toxicity with the use of ACE inhibitors has not been clearly defined, VASOTEC® (Enalapril Maleate, MSD) should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Postmarketing experience with all ACE inhibitors thus far suggests the following with regard to pregnancy outcome Inadvertent exposure limited to the first trimester of pregnancy has not been reported to affect fetal outcome adversely Fetal exposure during the second and third trimesters of pregnancy has been associated with fetal and neonatal morbidity and mortality

When ACE inhibitors are used during the later stages of pregnancy, there have been reports of hypotension and decreased renal perfusion in the newborn. Oligohydramnios in the mother has also been reported, presumably representing decreased renal function in the fetus. Infants exposed *in utero* to ACE inhibitors should be closely observed for hypotension, oliguria, and hyperkalemia If oliguria occurs, attention should be directed toward support of blood pressure and renal perfusion with the administration of fluids and pressors as appropriate Problems associated with prematurity such as patent ductus arteriosus have occurred in association with maternal use of ACE inhibitors, but it is not clear whether they are related to ACE inhibition, maternal hypertension, or the underlying prematurity

Nursing Mothers Milk in lactating rats contains radioactivity following administration of ^{14}C enalapril maleate. It is not known whether this drug is secreted in human milk Because many drugs are secreted in human milk, caution should be exercised when VASOTEC is given to a nursing mother.

Pediatric Use: Safety and effectiveness in children have not been established.

Adverse Reactions: VASOTEC has been evaluated for safety in more than 10,000 patients, including over 1000 patients treated for one year or more VASOTEC has been found to be generally well tolerated in controlled clinical trials involving 2987 patients.

HYPERTENSION The most frequent clinical adverse experiences in controlled trials were: headache (5.2%), dizziness (4.3%), and fatigue (3%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in controlled clinical trials were: diarrhea (1.4%), nausea (1 4%), rash (1.4%), cough (1.3%), orthostatic effects (1 2%), and asthenia (1.1%).

HEART FAILURE. The most frequent clinical adverse experiences in both controlled and uncontrolled trials were dizziness (7 9%), hypotension (6.7%), orthostatic effects (2.2%), syncope (2 2%), cough (2 2%), chest pain (2.1%), and diarrhea (2.1%).

Other adverse experiences occurring in greater than 1% of patients treated with VASOTEC in both controlled and uncontrolled clinical trials were: fatigue (1.8%), headache (1.8%), abdominal pain (1.6%), asthenia (1 6%), orthostatic hypotension (1 6%), vertigo (1.6%), angina pectoris (1.5%). nausea (1.3%), vomiting (1.3%), bronchitis (1.3%), dyspnea (1.3%), urinary tract infection (1.3%), rash (1.3%), and myocardial infarction (1.2%).

Other serious clinical adverse experiences occurring since the drug was marketed or adverse experiences occurring in 0.5% to 1% of patients with hypertension or heart failure in clinical trials in order of decreasing severity within each category

Cardiovascular Cardiac arrest; myocardial infarction or cerebrovascular accident, possibly secondary to excessive hypotension in high-risk patients (see WARNINGS, *Hypotension*); pulmonary embolism and infarction, pulmonary edema; rhythm disturbances, atrial fibrillation; palpitation.

Digestive Ileus, pancreatitis, hepatitis (hepatocellular or cholestatic jaundice), melena, anorexia, dyspepsia, constipation, glossitis, stomatitis, dry mouth.

Musculoskeletal. Muscle cramps.

Nervous/Psychiatric. Depression, confusion, ataxia, somnolence, insomnia, nervousness, paresthesia

Urogenital: Renal failure, oliguria, renal dysfunction (see PRECAUTIONS and DOSAGE AND ADMINISTRATION).

Respiratory: Bronchospasm, rhinorrhea, sore throat and hoarseness, asthma, upper respiratory infection.

Skin. Exfoliative dermatitis, toxic epidermal necrolysis, Stevens-Johnson syndrome, herpes zoster, erythema multiforme, urticaria, pruritus, alopecia, flushing, hyperhidrosis.

Special Senses. Blurred vision, taste alteration, anosmia, tinnitus, conjunctivitis, dry eyes, tearing

A symptom complex has been reported which may include a positive ANA, an elevated erythrocyte sedimentation rate, arthralgias/arthritis, myalgias, fever, serositis, vasculitis, leukocytosis, eosinophilia, photosensitivity, rash, and other dermatologic manifestations.

Angioedema: Angioedema has been reported in patients receiving VASOTEC (0.2%). Angioedema associated with laryngeal edema may be fatal. If angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with VASOTEC should be discontinued and appropriate therapy instituted immediately (See WARNINGS.)

Hypotension: In the hypertensive patients, hypotension occurred in 0.9% and syncope occurred in 0.5% of patients following the initial dose or during extended therapy Hypotension or syncope was a cause for discontinuation of therapy in 0 1% of hypertensive patients. In heart failure patients, hypotension occurred in 6.7% and syncope occurred in 2 2% of patients Hypotension or syncope was a cause for discontinuation of therapy in 1.9% of patients with heart failure. (See WARNINGS.)

Clinical Laboratory Test Findings.

Serum Electrolytes. Hyperkalemia (see PRECAUTIONS), hyponatremia

Creatinine, Blood Urea Nitrogen: In controlled clinical trials, minor increases in blood urea nitrogen and serum creatinine, reversible upon discontinuation of therapy, were observed in about 0 2% of patients with essential hypertension treated with VASOTEC alone. Increases are more likely to occur in patients receiving concomitant diuretics or in patients with renal artery stenosis. (See PRECAUTIONS.) In patients with heart failure who were also receiving diuretics with or without digitalis, increases in blood urea nitrogen or serum creatinine, usually reversible upon discontinuation of VASOTEC and/or other concomitant diuretic therapy, were observed in about 11% of patients. Increases in blood urea nitrogen or creatinine were a cause for discontinuation in 1.2% of patients.

Hemoglobin and Hematocrit. Small decreases in hemoglobin and hematocrit (mean decreases of approximately 0.3 g% and 1.0 vol %, respectively) occur frequently in either hypertension or heart failure patients treated with VASOTEC but are rarely of clinical importance unless another cause of anemia coexists. In clinical trials, less than 0.1% of patients discontinued therapy due to anemia

Other (Causal Relationship Unknown): In marketing experience, rare cases of neutropenia, thrombocytopenia, and bone marrow depression have been reported. A few cases of hemolysis have been reported in patients with G6PD deficiency

Liver Function Tests. Elevations of liver enzymes and/or serum bilirubin have occurred

Dosage and Administration: *Hypertension.* In patients who are currently being treated with a diuretic, symptomatic hypotension occasionally may occur following the initial dose of VASOTEC The diuretic should, if possible, be discontinued for two to three days before beginning therapy with VASOTEC to reduce the likelihood of hypotension. (See WARNINGS.) If the patient's blood pressure is not controlled with VASOTEC alone, diuretic therapy may be resumed

If the diuretic cannot be discontinued, an initial dose of 2.5 mg should be used under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions.*)

The recommended initial dose in patients not on diuretics is 5 mg once a day Dosage should be adjusted according to blood pressure response. The usual dosage range is 10 to 40 mg per day administered in a single dose or in two divided doses. In some patients treated once daily, the antihypertensive effect may diminish toward the end of the dosing interval. In such patients, an increase in dosage or twice-daily administration should be considered. If blood pressure is not controlled with VASOTEC alone, a diuretic may be added

Concomitant administration of VASOTEC with potassium supplements, potassium salt substitutes, or potassium-sparing diuretics may lead to increases of serum potassium (see PRECAUTIONS).

Dosage Adjustment in Hypertensive Patients with Renal Impairment: The usual dose of enalapril is recommended for patients with a creatinine clearance > 30 mL/min (serum creatinine of up to approximately 3 mg/dL). For patients with creatinine clearance ≤ 30 mL/min (serum creatinine ≥ 3 mg/dL), the first dose is 2 5 mg once daily The dosage may be titrated upward until blood pressure is controlled or to a maximum of 40 mg daily

Heart Failure. VASOTEC is indicated as adjunctive therapy with diuretics and digitalis. The recommended starting dose is 2.5 mg once or twice daily After the initial dose of VASOTEC, the patient should be observed under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. (See WARNINGS and PRECAUTIONS, *Drug Interactions.*) If possible, the dose of the diuretic should be reduced, which may diminish the likelihood of hypotension. The appearance of hypotension after the initial dose of VASOTEC does not preclude subsequent careful dose titration with the drug, following effective management of the hypotension. The usual therapeutic dosing range for the treatment of heart failure is 5 to 20 mg daily given in two divided doses. The maximum daily dose is 40 mg Once-daily dosing has been effective in a controlled study, but nearly all patients in this study were given 40 mg, the maximum recommended daily dose, and there has been much more experience with twice-daily dosing In addition, in a placebo-controlled study which demonstrated reduced mortality in patients with severe heart failure (NYHA Class IV), patients were treated with 2.5 to 40 mg per day of VASOTEC, almost always administered in two divided doses. (See CLINICAL PHARMACOLOGY, *Pharmacodynamics and Clinical Effects.*) Dosage may be adjusted depending upon clinical or hemodynamic response. (See WARNINGS.)

Dosage Adjustment in Patients with Heart Failure and Renal Impairment or Hyponatremia: In patients with heart failure who have hyponatremia (serum sodium < 130 mEq/L) or with serum creatinine >1.6 mg/dL, therapy should be initiated at 2.5 mg daily under close medical supervision. (See DOSAGE AND ADMINISTRATION, *Heart Failure*, WARNINGS, and PRECAUTIONS, *Drug Interactions.*) The dose may be increased to 2.5 mg b.i.d , then 5 mg b.i.d and higher as needed, usually at intervals of four days or more, if at the time of dosage adjustment there is not excessive hypotension or significant deterioration of renal function. The maximum daily dose is 40 mg

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VASOTEC is generally well tolerated and not characterized by certain undesirable effects associated with selected agents in other antihypertensive classes.

VASOTEC is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor.

A diminished antihypertensive effect toward the end of the dosing interval can occur in some patients.

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